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FOREWORD

FEW men at the age of sixty can look back with such satisfaction on their years of labor and count so many devoted friends, colleagues and inspired students all over the world as can Frank N. Wilson. His coming sixtieth birthday is a very happy occasion indeed. It gives us all an opportunity to express our great admiration and affection for him, feelings that so many have held but have never been able to make manifest in any outspoken fashion. Not many men in American medicine have occupied such a unique position as Dr. Wilson, for he has spent his life in an academic spirit of research, working both in the laboratory with animals and in the clinic with patients, constantly engaged in problems concerned with one subject, electrocardiography. Throughout this time he has been able to draw on two other disciplines, physics and mathematics. It was only after many years of labor that he could realize the practical results of his work and see them accepted and utilized throughout the world.

After completing his collegiate and medical education at the University of Michigan he quickly became interested in the new field of electrocardiography. At that time there was no outstanding figure working in this field in this country and so he had to depend upon his own talents. Although it was the custom in those days for recent graduates to work abroad in the clinic or laboratory of some celebrated physician, it is not surprising that Dr. Wilson was able to forego this advantage and make progress on his own. Not that he failed to derive inspiration and guidance from his teachers, such as Albion W. Hewlett, George Dock and later Sir Thomas Lewis. The outstanding fact is that in his chosen field of electrocardiography, which he made his life's work, one might call him a "self-made man."

In his early years his publications and his addresses at medical meetings met with little enthusiasm. Year after year he would appear at the Atlantic City meetings to present the results of his investigations. Generally the audience would simmer down to a faithful few who realized that his reports were vital and fundamental. Dr. Wilson never faltered in his efforts, convinced as he was of their importance and of their validity. Only after a decade or more did it become apparent to everyone that his series of investigations, both experimental and clinical, formed the main background upon which the electrocardiographic diagnosis of myocardial infarction rested. Only then did the clinicians realize that they had to listen to and to understand his work, for without it the care of their patients suffered.

It is historically of interest that Dr. Wilson began his researches on the ventricular complex just at the time Sir Thomas Lewis, who had been a pioneer in electrocardiography and had elucidated the subject of cardiac arrhythmias, had given up the study of electrocardiography thinking that there was little more to investigate, and had turned to the peripheral circulation for his interest. During the subsequent years Dr. Wilson clarified the entire subject of bundle branch block and the electrical changes resulting from myocardial infarction. In addition, he developed the method now widely used for taking unipolar leads and is to a large extent responsible for the increasing use of precordial leads.

The outstanding characterics of his contributions are their solidity and reliability. His clear mathematical mind gives his work the perma-

nence and accuracy that typifies the mother of all sciences. Long before an experiment was to be planned or some clinical observation was to be made, his mathematical analysis had foretold what was to be expected. A few years before any clinical observations were made on the precordial leads in cases of coronary thrombosis, he told one of his friends to put an electrode over the precordium and another one at any distant point and predicted that evidence of myocardial infarction would be found. Unfortunately, this wise suggestion was not heeded and a valuable discovery was delayed.

It is typical of the great investigator that when his work requires knowledge of a field somewhat different from and unrelated to his own, he plunges into it and acquires this information by his own talents. Most of us would stop short and restrict our researches to the limits imposed on them by our early training. Some time ago when Wilson was forced to take a sabbatical year because of his health, realizing that further knowledge of mathematics was necessary to prosecute some of his investigations, he literally plunged into this subject with youthful vigor. The year of convalescence proved very profitable. Not having had any formal instruction in higher mathematics during his undergraduate days, he was able to instruct himself in subjects of higher algebra, analytical geometry and the calculus with the aid of books and the refreshing atmosphere of his farm which he loves so much. Few men in medicine would have made such an attempt and fewer still would have succeeded.

This thoroughness and studiousness are well illustrated in his non-medical interests. Many years ago while working in the British Heart Hospital during the First World War, like many men in the service, he found a good deal of spare time on his hands. A fellow medical officer introduced the game of chess into the group and taught Wilson, who had never played the game, a few of the elementary principles. In a short while Wilson appeared with several books on chess and began studying them. Before long he became the master of the game and could outplay his teacher and the other members of the group. While in England working in close association with Sir Thomas Lewis whom he greatly admired, he developed an interest in bird photography, a hobby that Sir Thomas had enjoyed. This gave Wilson a great deal of joy during the subsequent years and enabled him to collect an elaborate variety of photographs of various birds and their nests and eggs. In this avocation he and his devoted wife have spent many happy days.

There is a lighter aspect of his character that only his intimates have had the privilege to know. Many friends have shared the rustic warmth of Wilson's farm. Here, one could see beyond the academic and erudite exterior a lively interest in wholesome pleasures. Worn clothes, new ideas, simple living, searching exchange of thoughts were part of the fare at the Stockbridge Farm. Nothing would delight him more than to have the group join in song around the piano and try out their lusty voices in singing favorite tunes. At these moments science was forgotten and art was in the ascendency.

Dr. Wilson has a faculty of gaining strong friendships, which have always meant a great deal to him. He has no interest in pomp or ceremony and prefers the solid virtues of truth and frankness. In this way he has endeared himself to many. The innumerable physicians, American and particularly foreign, who have visited him at his laboratory and worked with him, will ever feel indebted to him for his patience and his efforts, often extended at great personal sacrifice. It must be that he regards his function as a teacher and investigator to be a compelling motif.

Friends here and abroad extend their best wishes to him on his sixtieth birthday and to his dear wife who has so wisely aided him, and express their hitherto unspoken affection to him by publishing this *festschrift* in his honor.

SAMUEL A. LEVINE, M.D.

An Integrating Circuit for Measurement of the Areas of the Waves in the Electrocardiogram

By FRANKLIN D. JOHNSTON, M. D., RICHARD MCFEE, M. S., AND J. MARION BRYANT, M. D.

An electronic circuit capable of integrating the electrocardiogram is described. The net areas of the QRS complex, the T wave and of the entire ventricular complex, QRS-T, in the standard leads may be estimated by measurement of the length of two vertical lines in each of the integrated records. The manifest areas of QRS, T and the gradient (QRS-T) with the orientation of these vectors are easily calculated from the data. An example of an integrated electrocardiogram is reproduced, and the procedure for estimation of the gradient is presented.

INTRODUCTION

A QUICK and accurate method for the estimation of areas of certain deflections in the electrocardiogram has been needed since 1931 when Wilson, Macleod, and Barker[1] outlined the concept of the ventricular gradient and pointed out that it may be calculated if the net areas of the QRS complexes and T waves in any two of the standard limb leads are known. Various methods, all time consuming or subject to considerable error, have been used for the measurement of these areas, and we believe that the circuit to be described will be helpful to any physican or investigator who is interested in the ventricular gradient. An electronic integrator has been under investigation in this laboratory for several years, and recent simplifications have placed the outfit on a practical operating basis.

METHOD

The circuit employed for integration of the electrocardiogram is shown in Fig. 1. The integrator itself is diagrammed in detail and a great deal need not be said here concerning its construction or operation. When the push button switch (*S*) is pressed, the variable contact moves to the integrating position (*A*), the condenser, *C*, will charge, and a voltage $\left(E = \int \frac{idt}{C}\right)$ that is proportional to the integral of the electrocardiogram appears across its terminals. To record this voltage, as we have done, with a string galvanometer, the circuit must be arranged so that no current is drawn from the condenser, and this is the purpose of the double triode, the 6SL7. When the push button is not depressed the condenser, *C*, is short circuited, and a very small fraction of the output of the preamplifier is supplied to the input of the triode and on to the string. Thus, the string galvanometer records the electrocardiogram except when the integrating switch is depressed. The network of resistances with the battery is a balancing arrangement, and by proper adjustment of the coarse and fine controls the base line of the integrated curve (T–P interval) is made as nearly motionless as possible, before a record is actually taken.

Since vertical displacements in the integrated electrocardiogram are proportional to corresponding areas in the electrocardiogram itself, it is desirable, if possible, to record the two simultaneously. Most two-channel electrocardiographs may be used for this purpose, but we prefer to record the integral curve with the string galvanometer and have used the Cambridge Simplitrol Electrocardiograph with the mirror galvanometer (ordinarily used for sound tracings) to record the accompanying electrocardiogram. Since this galvanometer is rather insensitive, two double triodes, 6SN7, connected in parallel and arranged as indicated in figure 1, are employed to drive it.

The diagrams in figure 2 show in simplified form the type of records that are obtained and illustrate the way to measure the areas of QRS and T waves from the integrated record. In the ideal case shown in figure 2*A* the T–P intervals are horizontal, and the measurement is very simple. If the T–P intervals slant, as shown in figure 2*B*, due to imperfect balancing, the areas may be estimated again from the length of vertical lines, drawn in this instance from the slanting lines parallel with the T–P segment. Measurements in either situation will not give an accurate estimate of the areas unless complexes are selected where the inclination of the T–P intervals is essentially constant for several heart beats. We

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have used a transparent ruler with fine graduations ($\frac{1}{50}$ inch) to measure the vertical displacements

Ashman units by a simple standardization. The obvious way to accomplish this is to introduce into the

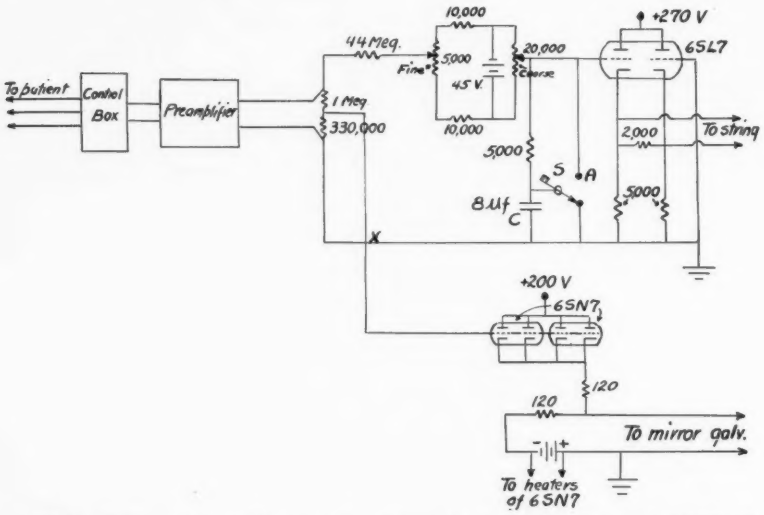

FIG. 1. Wiring diagram showing integrator and associated circuits. See text. NOTE: The wires should *not* be electrically connected at the point marked X. The high voltage supply for the 6SL7 should be +200 volts and not 270 volts.

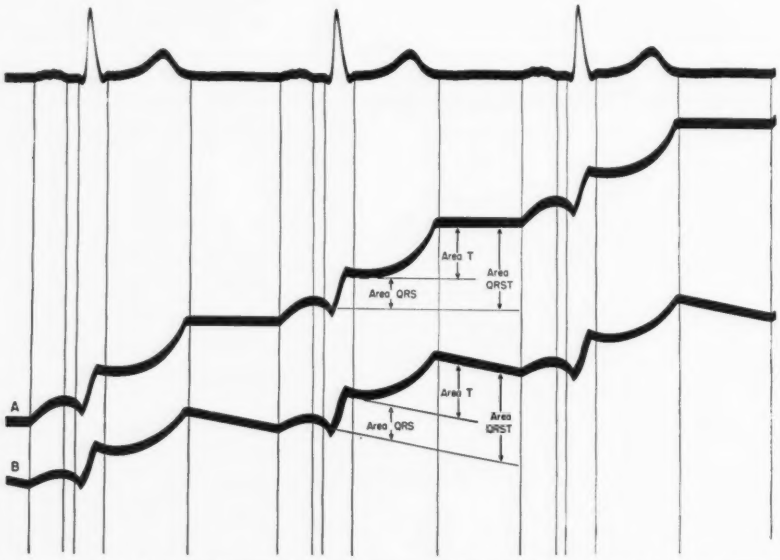

FIG. 2. Schematic diagrams illustrating how net areas of the QRS complex and the T wave may be obtained from integrated electrocardiograms. See text.

that represent the net areas of the QRS complex and the T wave, but these areas, expressed in arbitrary units, can be converted to microvolt seconds or

input of the preamplifier a fraction of a millivolt for a known period of time. This produces in the electrocardiographic record a known area, in microvolt

seconds, and the vertical displacement simultaneously observed in the integrated tracing is thus easily expressed in the desired units of area. It is possible to simplify the standardization procedure further. A constant ratio exists between the sensitivities of the circuit in the integrating (switch S depressed) and non-integrating positions. This ratio can be determined by experiment, and when it is known, the vertical displacements in the integrated curve are easily converted to areas in microvolt seconds. For our integrator the relationship is as follows:

$$\textit{Deflection per microvolt second (Integrated curve)} = \frac{\textit{Deflection per millivolt (Nonintegrated curve)}}{43}$$

This means that areas in microvolt seconds = K multiplied by the areas as measured in arbitrary units, where

$$K = \frac{43}{\textit{Deflection per millivolt (Nonintegrated Curve)}}$$

The preamplifier shown in figure 1 must have a voltage gain of approximately 50,000, very good stability, and be designed so it will amplify the electrocardiogram with reasonable fidelity.

Figure 3 illustrates the standard leads and the accompanying integrated records taken on a subject with a normal electrocardiogram. Reference is made to this figure and to table 1 in the description of the procedure, included below, for calculation of the gradient.

The lengths of the vertical lines labelled *A* and *B* represent the net areas of QRS and QRS-T in the three leads, and the actual measurements (made in 50ths of an inch) are indicated in the table in columns I, II and III. The figures for the areas of the T waves are gotten by subtracting the areas of QRS from those of QRS-T. Since Einthoven's law must be true for net areas over a given interval, as it is for instantaneous deflections, the figures representing the areas of QRS, T and QRS-T in Lead II should equal the algebraic sum of those in Leads I and III. Due to errors of measurement (usually caused by uncertainties regarding the position of the baseline), the calculations do not ordinarily check as they should, and in order to minimize and distribute these errors, the figures are adjusted by a simple calculation. The correction to be applied to each of the areas is very easily gotten, and the adjusted areas may be defined as follows:

$$I_a = I - 1/3\,\Delta$$
$$II_a = II + 1/3\,\Delta$$
$$III_a = III - 1/3\,\Delta$$
$$\textit{where } \Delta = I + III - II$$

TABLE 1

COMPLEX	I	II	III	Δ	I_a	II_a	III_a
QRS	11.7	18.0	8.7	2.4	10.9	18.8	7.9
QRS-T	26.0	37.0	14.7	3.7	24.8	38.2	13.5
T	14.3	19.0	6.0	1.3	13.9	19.4	5.6

COMPLEX	$2II_a - I_a$	V	H	$\sqrt{V^2 + H^2}$	$\alpha°$	M(μvs)	M(units)
QRS	26.7	15.4	10.9	18.9	54.6	34.6	8.7
QRS-T	51.6	29.8	24.8	38.8	50.1	71.0	17.8
T	24.9	14.4	13.9	20.0	46.0	36.6	9.2

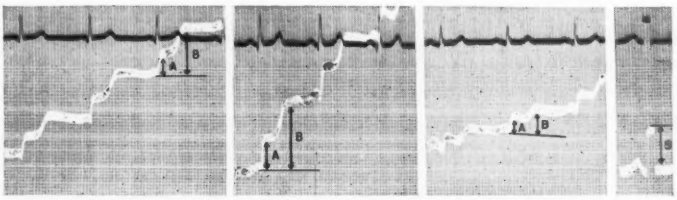

FIG. 3. An illustration of integrated electrocardiograms taken with the arrangement described. The standardization record is mounted to the right of the limb leads. See text.

The values for Δ are given in the table, and the adjusted areas are listed in the appropriate columns. It will be observed that the adjusted areas for QRS, QRS-T and T fulfill Einthoven's law.

When the adjusted areas are available, calculations of the manifest value of QRS, T and QRS-T and the direction of these vectors (angle α) may be made in several different ways, following the principles that have been used for years for estimation of the manifest electrical vector at any instant of time. We have done this by determining the horizontal (H) and vertical (V) components of the vector in question and then by using the following expressions to determine the magnitude of the force and its orientation:

$$\textit{Magnitude} = \sqrt{H^2 + V^2}$$

$$Direction\ (angle\ \alpha) = arctan\ \frac{V}{H}$$

$$where\ H = I_a\ \ and\ \ V = \frac{II_a + III_a}{\sqrt{3}} = \frac{2II_a - I_a}{\sqrt{3}}$$

These quantities have been calculated and are shown in the table. The values for the angle α are independent of units used in measurement of the areas and need no adjustment. To express the magnitude of the vectors in microvolt-seconds instead of arbitrary units of area, the method outlined in an earlier paragraph is employed. The factor, K, by which the areas must be multiplied to convert them to microvolt-seconds is here 43/23.5 = 1.83, where 43 is a constant of the integrating circuit determined by experiment and 23.5 is the deflection of the string shadow when 1 millivolt is introduced into the input circuit of the pre-amplifier and the integrating button is in the neutral (non-integrating) position. This

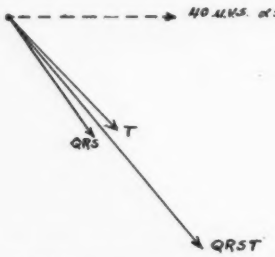

FIG. 4. Graphic representation of the vectors QRS, QRS-T and T for the electrocardiogram presented in this article. See table 1 and text.

standardization is shown in figure 3 with the limb leads. The magnitudes of the vectors QRS, QRS-T and T expressed in microvolt-seconds and also in Ashman units $\left(\frac{\text{microvolt-seconds}}{4}\right)$ will be found in the table. These vectors are shown graphically in figure 4. They are plotted with reference to a horizontal line (angle $\alpha = 0°$) and their size is proportional to the dotted horizontal line which represents 40 microvolt-seconds (10 Ashman units).

DISCUSSION

The circuit and technic described above for estimation of the gradient have been employed with over one hundred patients, and we are satisfied that the method saves enough time and is sufficiently accurate to justify its use in a laboratory where it is desired to determine the gradient for a large number of electrocardiograms.

The most troublesome problem that arises, irrespective of the method one may use to estimate the net areas of QRS and T, is to decide where the baseline should be located. Careful inspection of many electrocardiograms reveals that the baseline (T–P interval) is not a straight horizontal line, and in the integrated records these difficulties are multiplied. We have no ready solution for this problem but think some generally accepted arbitrary method for placing the baseline may be necessary. Another situation that often complicates the use of the integrator arises from slow fluctuations of the "skin potentials." It is usually easy, with the integrating circuit we have described, to adjust the balancing resistances so that the T–P intervals of the integrated record are represented by horizontal or nearly horizontal segments for a few cycles but rarely does this baseline retain a horizontal or other constant position for more than a few seconds at a time.

The foregoing difficulty might be minimized by the use of a preamplifier with a time constant sufficiently long to pass the low frequency components of the electrocardiogram without significant attenuation and short enough to block out very low frequency drifts that may lead to fluctuations of the baseline. There are objections, however, to the use of a resistance-condenser coupled pre-amplifier, particularly one with a relatively short time constant, and at the moment we are using a differential amplifier capable of direct current amplification designed by one of the authors (Richard McFee). Incidentally, after the apparatus has been turned on for a sufficient period (fifteen to thirty minutes), there is great stability, and drift due to the pre-amplifier and associated circuits is negligible.

Because of the chance that part, at least, of the variable "skin currents" that have proved so troublesome to us are not due to variable electromotive forces arising beneath the surface of the skin but are due to slight differences between the electrodes, we are planning to investigate this possibility.* Should the use of

* An electrical engineer who has designed a cathode-ray electrocardiograph with a D.C. differential amplifier has been able to practically eliminate low frequency drifts by the use of special electrodes. The

special electrodes eliminate all or most of the low frequency drift, the value of the integrator we have described will be considerably enhanced.

Conclusions

1. A circuit for integration of the electrocardiogram is described, and the method by which the gradient may be estimated is outlined in detail.

2. Low frequency shifts of the baseline which are not constant for any great period of time are the most troublesome problem encountered in the use of the apparatus. Special electrodes may aid in the elimination of this difficulty.

authors, unfortunately, cannot remember the name and address of this individual but wish to give him credit for the idea.

REFERENCE

[1] Wilson, F. N., Macleod, A. G., and Barker, P. S.: The T deflection of the electrocardiogram. Tr. A. Am. Physicians. **46:** 29, 1931.

Intracardiac and Intravascular Potentials Resulting from Electrical Activity of the Normal Human Heart

By CHARLES E. KOSSMANN, M. D., ADOLPH R. BERGER, M. D., BERTHA RADER, M. D., JOSEPH BRUMLÍK, M. D., STANLEY A. BRILLER, M. D., AND JOHN H. DONNELLY, M. D.

Electrical records were made from the interior of the right heart and attached vessels in 14 normal subjects. Technics differed somewhat from those previously reported in that simultaneously recording string galvanometers were used exclusively, and tracings were made whenever the catheter-electrode was moved within the heart. Previously unreported large positive QRS deflections were found in records from the lower part of the right atrium, from the pulmonary artery, and in two instances from within the right ventricle itself close to the base. The possible anatomical origin of these is discussed.

SEVERAL papers[1–10] are available on the nature of the electrical records obtained from the interior of the human heart. The additional verbal reports being made with increasing frequency attest to the interest in the possible answers these records may supply to many questions in fundamental electrocardiographic theory.

Intracardiac leads in man were begun in this laboratory in 1946. This report is concerned with the findings in 14 subjects with normal hearts as determined clinically. Emphasis will be placed on those aspects of our studies which differ from or supplement those previously reported.

METHODS

The catheters* used were 100 cm. long and ranged in caliber from 6 F to 9 F. They consisted of a core of fine copper wire connected at the distal end to a silver electrode and at the proximal end to an ordinary electrical plug. All of the wire and the proximal, flanged end of the electrode were encased in specially impregnated radiopaque nylon or silk. The exposed tip of the electrode was 3 mm. in length. In early experiments this was coated with silver chloride but the coating was later discovered to be unnecessary with the electrical circuit used in recording. "Springiness" of the catheter could be modified by changing the caliber of the copper wire core.

Actual introduction of the catheter into the heart was by technics previously described.[9–11] We have found the left median basilic vein the best point for insertion for two reasons: (1) the curve described by the left subclavian and innominate veins, the superior vena cava, and the right atrium seems to favor easy entrance of the electrode into the right ventricle and pulmonary artery; (2) an opportunity is afforded to explore the interior of the thorax along the veins mentioned.

Electrocardiograms were made with two Hindle string galvanometers* recording simultaneously. In circuit with the first (upper string shadow in all illustrations) was an originally designed single stage amplifier with balanced plate circuit. This amplifier, which maintained an output impedance match of 2000 ohms with the string at all times, had a frequency response which was flat from 0 to 100 cps when used with the string under conditions simulating those of the experiments (Fig. 1).† No reso-

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This study was aided by grants from the New York Heart Association and the Knapp Foundation.

* Manufactured according to specifications by the U. S. Catheter and Instrument Company, Glens Falls, New York.

* Cambridge Instrument Co. Models No. 2 and No. 3.

† This desirable frequency feature of our recording instrument may explain some of the differences in our records when compared with those previously published. The latter, with few exceptions[4, 5, 7, 8] have been made with oscillographic types of instruments the sinusoid voltage response of which is known to be 100 per cent of the equivalent square wave response only between 12.5 and 25 cps. The former is the minimum natural frequence of an electrocardiograph defined in a somewhat different way by Lewis and Gilder[13] although it is noteworthy that all of Lewis' experiments were done with an instrument the

nant peaks were discovered following the input of sinusoid voltages up to 300 cps. Actually only the last ten patients were studied with this circuit. With the first four (JRu, JNe, MSh, and JCo) an amplifier with less desirable characteristics was used. Its principal defect was a resonant peak in the range 50 to 60 cps. This occasionally resulted in considerable exaggeration of the notch on the down-stroke of S sometimes encountered in leads from the base of the right ventricle. This was easily recognized. Such deflections were omitted from the few calculations made on the leads of these four patients.

The second electrocardiograph was used in the ordinary way to record a variety of external leads as desired (lower string shadow in all illustrations). The extremity, precordial (Leads V_1 to V_6), external thoracic (Leads V_8, V_B, V_{4R}, V_{6R}, and sometimes others*) and intracardiac potentials were obtained by connecting one end of the string to the central terminal of Wilson, Macleod, and Barker.[18] The circuit was arranged to augment extremity potentials.[19] The galvanometric connections were so made that in the finished tracing a downward deflection represented relative negativity of the exploring electrode.

The string sensitivity used was quite variable, especially for the endocardial leads, and is indicated on each illustration.

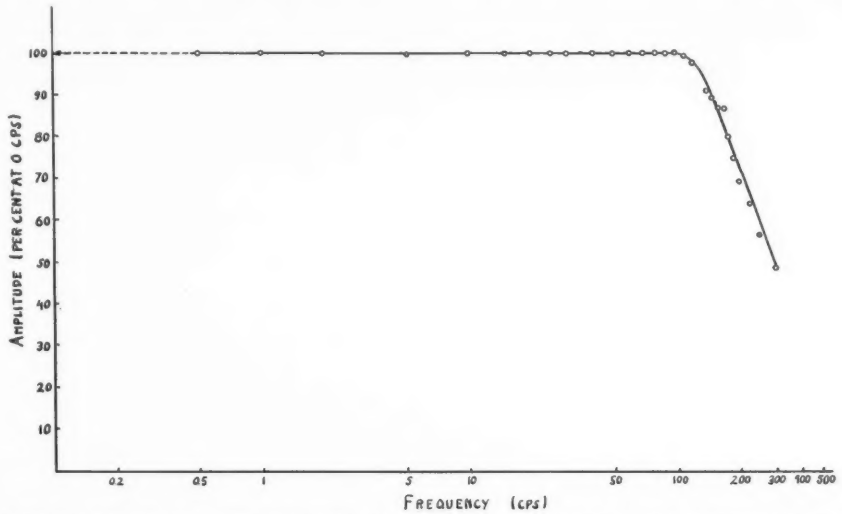

FIG. 1.—Amplitude-frequency characteristics of the galvanometer used with an amplifier to record the intracardiovascular potentials. String tension was regulated to simulate usual experimental conditions (1 mv. = 0.5 cm.). Constancy of voltage output (3.4 mv.) of the oscillator (Hewlett Packard Model 202B) was checked with a Dumont oscilloscope type 250 utilizing DC amplifier only, and corrections made if deviations occurred.

natural frequency of which was "approximately 100–200 (cycles) per second under ordinary working conditions."[14] Einthoven[15] specified that an instrument having a rise time (deflection time) equal to 0.01 second for a 1 cm. rise (equivalent to a frequency of 25 cps) would have an adequate frequency bandwidth for cardiographic recording. Holzer,[16] on the basis of studies on standard leads, has established a flat frequency response to 200 cps as a requirement of an instrument. Reid and Caldwell,[17] by a Fourier analysis of normal standard leads, discovered some important frequency components up to 125 cps.

We have observed intracardiac deflections with a frequency in excess of 60 cps with the string galvanometer and amplifier used. Such deflections will be obscured or completely unrecorded by the oscillographic instruments referred to, and others will be exaggerated if resonant peaks exist in the frequency response curve. The ideal recorder for intracardiac potentials is not certain but from these data it would seem undesirable to have its natural period longer than 0.01 second.

At first the intracardiac potentials were recorded from random locations in the heart. As teamwork improved we attempted to place the electrode initially in the pulmonary artery or one of its branches, usually the right. Its

* Chest position 8 was in the left midscapular line; B, in the midline posteriorly. Both were at the same horizontal level as position 5.

frontal projection was determined with the central ray of the fluoroscope and the point was marked on the chest. It was customary to record this intra-arterial potential first with Lead I, then with a precordial lead when the

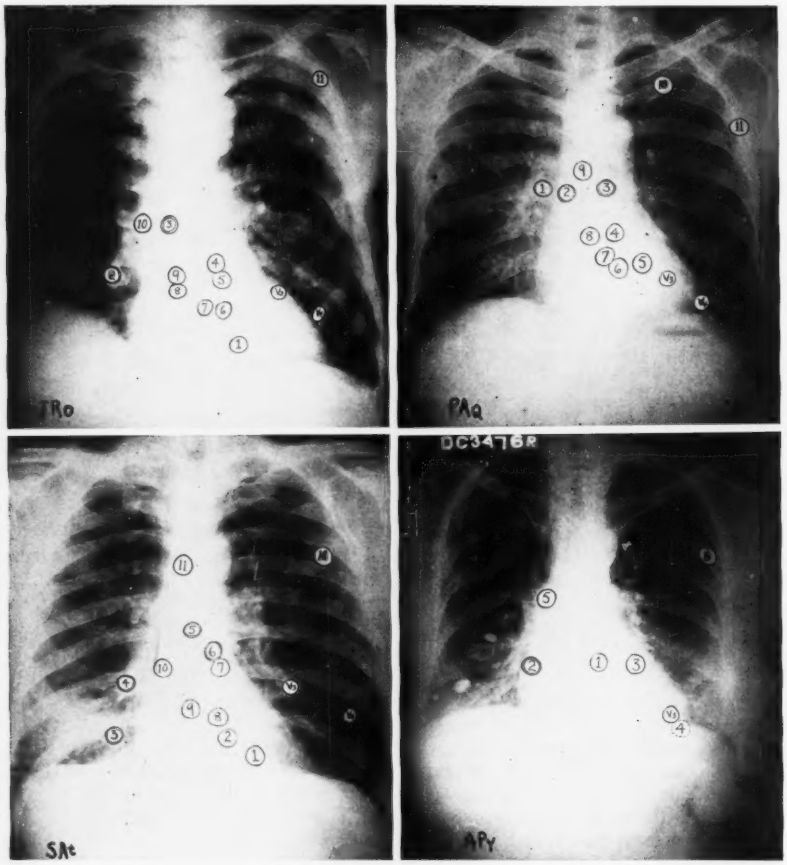

FIG. 2.—Teleroentgenograms of four normal subjects. The identifying initials are in the lower left hand corner of each illustration. The single circles with a figure within indicate the approximate location of the intracardiac electrode at the time the electrocardiographic record was made. A double circle with a figure enclosed indicates that the electrode was in the pulmonary artery or its right branch. In patient APy, point 4 was directly under precordial lead V_3. In each instance the location of the electrode when recording precordial leads V_3 and V_4 is shown for comparison.

exploring electrode (3 cm. in diameter) on the chest was directly over the internal electrode. Thereafter the electrode was slowly withdrawn several centimeters as a continuous electrocardiographic record was being made. The new location was checked fluoroscopically, records were made again with Lead I and with a precordial lead, and this whole procedure repeated. By this means it was usually possible to explore quite thoroughly the right pulmonary artery; the pulmonary stem; the conus, outflow tract, apex, inflow tract and tricuspid region of the right ventricle; all levels of the right atrium; the superior vena cava; and the left innominate, subclavian and axillary veins. In addition, it was possible to obtain records during transit between the pulmonary artery and right ventricle, the right ventricle and right atrium, and the right atrium and superior vena cava. Although

it was usually easy to identify such transitions electrically, there were occasions when pressure tracings would have provided additional reassuring information.

Tracings were particularly difficult to make when the electrode was in a vein. The string shadow was often distorted by fine oscillations of such type as probably came from electrical activity of the muscle in the venous walls.

TABLE 1.—*Pertinent Data on 14 Patients With Clinically Normal Heart*

Patients	Age	Sex	Color*	Diagnosis
JRu	70	M	W	Subdeltoid bursitis
JNe	55	M	N	Pituitary neoplasm
MSh	45	M	N	Lobar pneumonia, type XII, convalescent
JCo	49	M	W	Lobar pneumonia, type VI, convalescent
JJa	56	M	W	Psychoneurosis; latent syphilis
JMa	43	M	W	Multiple sclerosis
APy	69	F	W	Bronchopneumonia, convalescent
WLCa	53	M	N	Grippe, convalescent
PAq	49	M	W	Grippe, convalescent; latent syphilis
SAt	58	M	W	Pneumococcus pneumonia and meningitis, convalescent
QWi	44	M	N	Post-traumatic epilepsy
EFr	49	M	N	Cerebrovascular syphilis
JPHe	52	M	W	Intermittent anomalous A-V excitation; lobar pneumonia, convalescent
JRo	61	M	W	Adenocarcinoma of prostate.

* White or Negro.

We have frequently encountered the positive displacement of the RS-T segment and the accompanying distortion of QRS which results from pressure of the intracardiac electrode on the endocardium.[20] Comparable modifications have also been seen in the atrial deflections. Usually such distortion of the tracing can be recognized; rarely it is difficult to detect. Unfortunately, distorted records of this kind have already appeared in the literature as representative of normal endocardial potentials.

After the completion of an experiment, lead markers were placed on the skin at the various locations of the intracardiac electrode. A teleroentgenogram was then made, in the beginning with the patient first recumbent and again standing, but later only standing. The reason for this was that little difference was found in the location of the markers on the x-ray films made in the two positions. Further, the films obtained with the patient standing were far better, and displacement of the markers was less likely to occur (Fig. 2).

The clinical data pertinent to the 14 patients included in this report are incorporated in table 1.

All measurements were made with the Elliott comparator manufactured by the Cambridge Instrument Company. The error of measurement with this device, when interindividual variations are excluded, is less than ±0.003 second.

Records from Within The Pulmonary Artery

In half of the 14 normal subjects studied, the exploring electrode could be placed in the pulmonary artery or its right branch. The records obtained during electrical activity of the atria and of the ventricles were quite variable as might be expected from the considerable length of the tributaries, and the variable relations particularly of the right branch to the different chambers of the heart. These relations are summarized in the following paragraph from Morris' Anatomy.[21] The italics have been put in by us to emphasize the relation of the pulmonary artery and its right branch to the left atrium, and of the former to the left auricle as well.

Anatomical Relations of the Pulmonary Artery and the Right Pulmonary Artery

The pulmonary artery has a course of approximately 5 cm. within the pericardium before dividing into the right and left branches. In front it is covered by the remains of the thymus gland and the pericardium. Behind it lies successively upon the ascending aorta and the *left atrium*. To the right are the ascending aorta, the *right atrium*, the right coronary artery, and the cardiac nerves. To the left are the pericardium, the left pleura and lung, the *left auricle*, the left coronary artery, and the cardiac nerves.

The right pulmonary artery passes almost horizontally under the arch of the aorta to the root of

the right lung where it divides into three branches, one for each lobe. In its course to the lung it has in front of it the ascending aorta, the superior vena cava, the phrenic nerve, the anterior pulmonary plexus, and the reflection of the pleura. Behind are the right bronchus and the termination of the azygos vein. Above is the arch of the aorta, and below are the *left atrium* and the upper right pulmonary vein.

The nature of the *atrial deflections* observed in the right pulmonary branch depended on whether the exploring electrode was low or high in this vessel or one of its main tributaries.

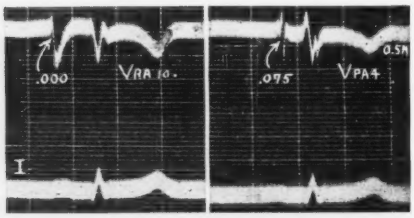

FIG. 3.—Patient JPHe. On the left above is a record made from a point high in the right atrium ($V_{RA_{10}}$), and on the right above a record made from within the pulmonary artery (V_{PA_4}) just above the pulmonic valve. Both were made at the same string sensitivity (1 mv. = 0.5 cm.), and both with the same simultaneous lead (I, lower records). Features of the P wave are to be noted, particularly the small Ra and the broad slurred Sa in the lead from the right atrium, and the notched, slurred Ra and sharp, brief Sa in the lead from the pulmonary artery. The figures indicate the time at which the intrinsic deflections crossed the baseline, regarding the first as zero. The figure of 0.075 sec. obtained in the lead from the pulmonary artery is ascribed to the late excitation of the left auricle (appendage) which is close to the electrode in this location. Time lines occur every 0.2 sec.

When the electrode was low the initial part of the accession process[22] consisted of a moderately late positive deflection ("extrinsic deflection" of Lewis,[23] "e" of Hurst Brown,[24] "a_1" of Macleod[22]); when the electrode was high the principal part of the accession process was negative, and the extrinsic deflection terminated early. This is approximately what one would expect in the light of existing theories if the electrode were close to the sinoatrial node when high in the pulmonary artery, and close to the inferior end of the right atrium when low in one of its tributaries.

In the pulmonary artery the initial atrial deflections varied greatly from predominantly negative to predominantly positive. In the one latter instance (patient JPHe, Fig. 3) the end of an easily recognized extrinsic deflection was quite late, almost at the end of the P wave in Lead I, suggesting that the electrode was being affected by the left atrium or auricle (appendage) which are in close proximity to the pulmonary artery.

The nature of the *ventricular deflections* varied also. In the right branch these were usually similar to comparable deflections in the lead from the right arm, with minor variations. The combinations observed were rSr′, QR (with slurred, broad R); QS (with one or several notches), or rS. The T wave in all of these was inverted.

In the pulmonary artery the combinations observed were rSr′, rS (with S notched), qRs, rsr′s′, and rsR′. The complex, rSr′, when present, simulated the initial ventricular deflections in Lead aV_R (and presumably in Lead V_R); rS simulated what was found in leads from the greater part of the right ventricular cavity. The qRs was not unlike the ventricular deflections recorded in the same patient (SAt, Fig. 9) from the lower part of the right atrium, and rsR′ in the one patient in which it was encountered (patient APy, Fig. 4) was similar to potentials recorded from the conus and the tricuspid areas of the right ventricle, and from the lowest part of the right atrium (see below). In the one instance (patient PAq, Fig. 5) of a quadriphasic record (rsr′s′) unusually high positive QRS deflections were encountered in the tricuspid region of the right ventricle, and in the lower part of the right atrium. Further reference will be made to these important positive deflections later. The T wave in all the records from the pulmonary artery was inverted.

These data may be summarized as follows: the nature of the galvanometric deflections encountered as a result of action currents in atrial and ventricular muscle are more variable when recorded from the main stem than from the right branch of the pulmonary artery. In the former the atrial deflections are apparently determined by the relative closeness of the

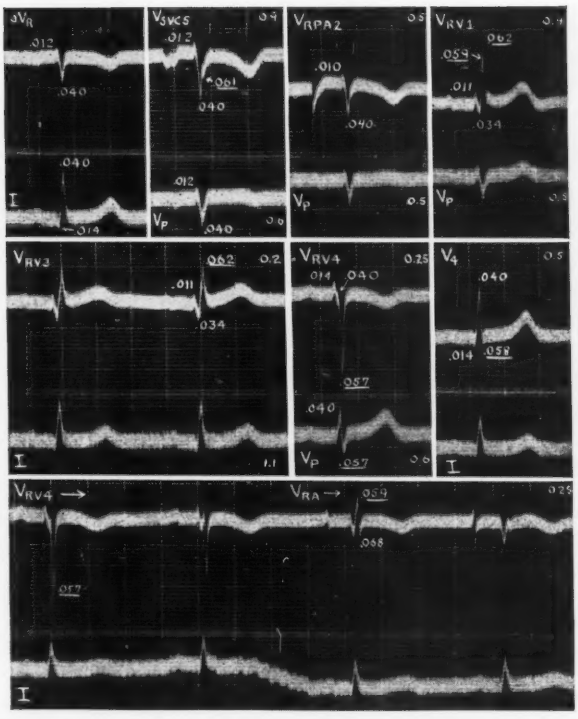

FIG. 4.—Patient APy. A variety of internal and external leads recorded simultaneously with Lead I (I), or with a lead (V_P) taken with a precordial electrode directly over the intracardiovascular electrode. The records in the first two rows have been arranged approximately in the order of their occurrence as a line is followed on or in the thorax from the right arm to the left side of the precordium. Each lead is indicated by the symbol V followed by a subscript of letters and a number. The letters indicate the structure from which the lead was made. The number corresponds to the number of the intracardiac point as seen in figure 2. For example: V_{SVC5} means potential of the superior vena cava at point 5; V_{RPA2}, potential of the right pulmonary artery at point 2; V_{RV1}, potential of the right ventricle at point 1; V_{RA}, potential of the right atrium at an unknown location. The number written at the right end of each trace denotes the sensitivity of the string at which it was recorded. If left blank it means that the sensitivity of the string was normal (1 mv. = 1 cm.). Time lines occur every 0.2 sec.

The remaining numbers on the records indicate the time of the adjacent QRS deflection with respect to the beginning of QRS in Lead I. Those waves which are simultaneous with the large positive deflection encountered in leads V_{RV1} and V_{RV3} at the base of the right ventricle are underlined. In contrast to records of other patients (Figs. 5 and 6) the wave under consideration in lead V_{SVC6} is a negative notch on the ascending limb of S, and cannot be identified at all in lead aV_R. The intraventricular leads differ from others, too, in the occurrence of a positive T wave in leads V_{RV1} and V_{RV3}.

The lowest row shows a continuous record made simultaneously with Lead I as the electrode was withdrawn rather rapidly from the apex of the right ventricle through the tricuspid orifice into the right atrium. The time of the S wave at the apex of the right ventricle (first complex) is almost identical with the time of the positive deflection seen as soon as the electrode has entered the right atrium (third complex). Location of the electrode in the latter chamber at the time is deduced from the initial occurrence of a diphasic P wave in the record.

upper part of the right atrium or auricle on the one hand and the left atrium or auricle on the other. The initial ventricular deflections may simulate those seen in the lead from the right branch and hence the lead from the right arm. However, in three of seven instances late

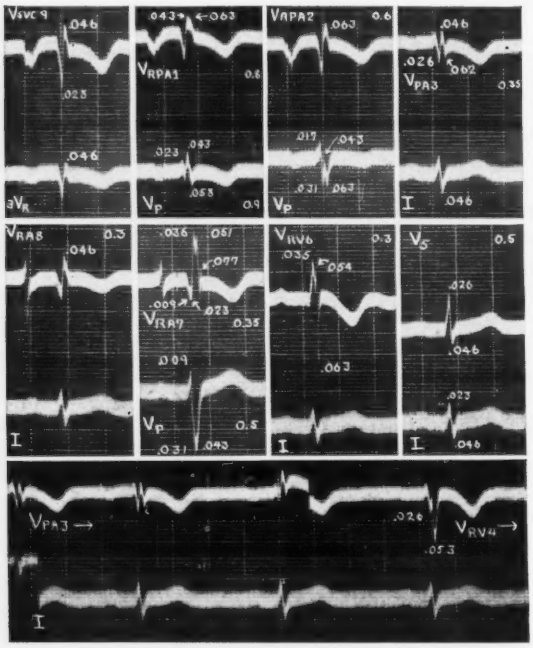

FIG. 5.—Patient PAq. Records have been arranged much as in Fig. 4, and symbols and figures have the same significance.

In this record the large positive deflection in the lower right atrium (V_{RA7}) and in the tricuspid region of the right ventricle (V_{RV6}) consists of two peaks, 0.036 and 0.051 sec. after the beginning of QRS in Lead I. The analog in vascular and surface leads (aV_R, V_{SVC9}, V_{RPA1}, V_{RPA2}, V_{PA3}, V_{RA8}, and V_5) has a time of 0.046 or approximately the mean of the previous two figures.

To be noted is that the deflection in question in a precordial lead (V_P) over point 1 (V_{RPA1}) is positive and over point 2 (V_{RPA2}) it is a positive notch on the S wave, but in leads farther to the left (V_P of V_{PA3}, not shown, and V_{RA7}) it is negative. Reference to Fig. 2 will demonstrate that points 1 and 2 are quite far to the right at the level of the third rib, while the remaining points may be regarded as closer to the region of the pulmonic conus.

The small deflection in the middle of the P-R segment of lead V_{RA7} is probably a second intracardiac deflection with origin in a remote part of the atrium (see Fig. 10).

The lowest row shows a continuous record made as the electrode was withdrawn from the pulmonary artery (V_{PA3}) into the conus (V_{RV4}, last complex). Standardizations (1 mv. = 0.4 cm.) distort the third complex. The change of the late positive to a completely negative deflection is easily seen. The simultaneous lead is Lead I.

positive QRS deflections of relatively large size were encountered, and these were usually accompanied in the same subject by similar late deflections in leads from the interior of the base of the right ventricle or from inferior levels of the right atrium, and had recognizable counterparts in external leads to be considered below.

PHENOMENA OBSERVED WITH CONTINUOUS RECORDING WHILE MOVING THE ELECTRODE FROM THE PULMONARY ARTERY TO THE CONUS OF THE RIGHT VENTRICLE

In six instances a record of the intracardiovascular potential was made simultaneously with Lead I as the electrode was slowly withdrawn from the pulmonary artery to the pulmonary conus of the right ventricle. The change observed was sometimes abrupt, sometimes gradual. In the latter instance it was not possible to say exactly when the electrode had entered the right ventricle.

The changes seen were those involving the P wave, the QRS-T, premature systoles, the development of an ectopic rhythm, and the occurrence of S-T elevation as the electrode made pressure on the ventricular endocardium.

If the P wave was originally inverted in the

pulmonary artery, it became shallower, isoelectric or slightly diphasic in the right ventricle. If it was diphasic to begin with, the wave would become isoelectric.

If the QRS was originally the rS or rSr′ type, the r would become more prominent, the S deeper, and the r′ would become a negative peak on the ascending limb of S. T also became deeper. The voltage of all deflections was quite variable (Table 2) but in this transition (pulmonary artery to conus) it increased approximately twofold.

In two instances the change in QRS was quite striking in that a large positive deflection appeared in the ventricle with a positive T wave. One of these patients displayed anomalous atrioventricular excitation[25] at times; the other was a woman, 69 years old, but with no clinical or electrocardiographic evidence of heart disease (Fig. 4). The composition of QRS in these differed one from the other. In the first it was qRS and in the second it was rsR′.

In four of the six instances a characteristic change in P and QRS occurred at the same time as the electrode entered the right ventricle; in one instance all deflections were obscured by the development of an irregular ventricular rhythm; and in another the exact time of entry of the electrode into the ventricle could not be determined electrically.

Atrial premature systoles were observed once during movement of the electrode. It is likely that they were produced by pressure of proximal parts of the catheter in the right atrium. An irregular ectopic rhythm and premature systoles of ventricular origin were each seen once. In the former instance when the rhythm stopped spontaneously the intraventricular action potential was found to be distorted by an elevated S-T segment ("injury potential,"[26,27] "demarcation potential"[28]).

Records from within the Right Ventricle

From the first few experiments in which the electrode was placed in random fashion in the normal right ventricular cavity, it was our impression, as reported by others[2–4, 7, 10], that the right intraventricular deflections during ventricular excitation consist of a small R, a deep S, and an inverted T wave. Some notching or slurring of either of the first two deflections occurred, and it appeared that only minor variations in this record resulted from placement of the electrode in different parts of the chamber. It was only when we began to use a technic of continuous recording as the electrode was moved either within a vessel or chamber, or from a vessel to a chamber and the reverse, that considerable variation in the records was encountered, particularly at the base of the right ventricle in close proximity to either the pulmonic valve or the tricuspid valve. These may be seen in Figures 4, 5, and 6.

With two exceptions the initial ventricular deflection in the right ventricle was positive.

TABLE 2.—*Size of the RS (or QR) Deflection in Various Leads of 14 Patients without Heart Disease as an Index of the Relative Size of the Voltages Developed within the Right Heart and at the Surface of the Body (in tenths of a millivolt).*

Lead	I	II	III	aV_R	aV_L	aV_F	V_1	V_2	V_3	V_4	V_5	V_6	V_E	V_{3R}	V_{8R}
Min.	2.0	4.0	2.0	1.9	0	1.9	4.0	6.0	10.0	14.0	9.0	6.0	4.0	2.0	2.0
Max.	14.3	36.0	13.0	18.0	10.0	17.0	38.0	50.0	42.0	44.0	28.0	24.0	54.0	20.0	10.0
Mean	7.5	11.7	6.7	9.0	4.4	7.7	14.2	22.0	21.0	25.5	18.3	11.2	18.0	7.2	4.2

Lead	Superior Vena Cava	Right Pulm. Artery	Pulm. Artery	Right Atrium			Right Ventricle					Left Axillary Vein
				High	Mid	Low	Conus	Outflow Tract	Apex	Inflow Tract	Tricuspid Region	
Min.	4.0	7.0	12.5	8.0	9.0	13.0	24.0	43.0	36.0	26.0	26.0	2.0
Max.	23.3	20.0	28.0	26.0	30.0	29.0	90.0	120.0	230.0	200.0	47.5	8.0
Mean	13.8	13.3	21.3	16.6	18.7	17.8	48.1	71.8	100.5	66.5	33.0	5.2

The size of this R wave varied in different parts of the chamber. In some instances it was larger at the base than at the apex; in others the reverse was true. Between the region of the conus and the region of the tricuspid valve there were often considerable differences in record was not different from what was seen in the other patients.

The initial positive deflection has been ascribed to early depolarization of the left side of the interventricular septum.[14] In general the area concerned is believed to be near the

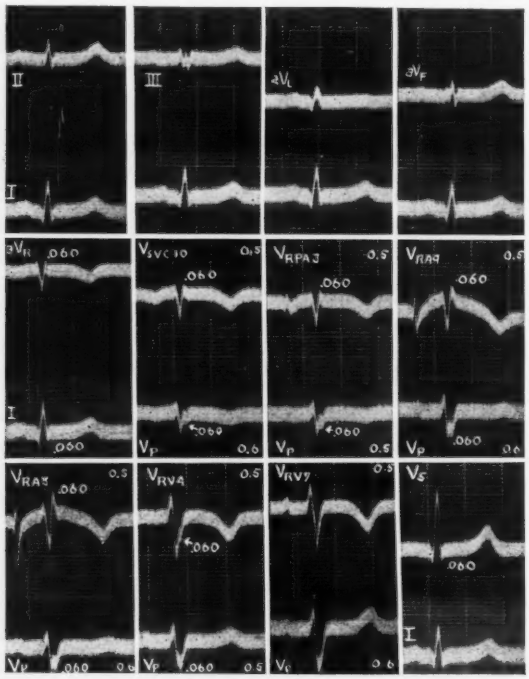

FIG. 6—Patient JRo. A variety of leads arranged as in Figures 4 and 5 in the lower two rows. The upper row shows the standard leads and the augmented extremity potentials (I, II, III, aV_R, aV_L, aV_F). The simultaneous lead is Lead I or a point on the precordium (V_P) over the internal electrode. In this figure only the deflections simultaneous with the late positive deflection in the atrium (V_{RA8} and V_{RA9}) are labelled with the figure 0.060, the time in seconds it occurred after the beginning of QRS in Lead I. In this instance the deflection in question was negative in all leads below the tricuspid orifice and on the front of the chest; it was positive in leads from the atrium, the superior vena cava, the pulmonary artery (not shown) and its right branch, and the right arm. Times of other QRS deflections may be found in Table III. The initial Qa wave in the auricular complexes of leads V_{RA8} and V_{RA9} preceded the onset of the P wave in Lead I by 0.002 sec.

this deflection. When present, it was usually slurred or notched.

The two exceptions were patients PAq and JPHe. The former showed a depression preceding a large, late, notched R when the electrode was in the tricuspid orifice (Fig. 5, V_{RV6}); the latter, already referred to, displayed a Q wave in leads from the pulmonary conus and from the apex but in the tricuspid region the base of this structure; the findings we have presented neither support nor deny this. The variables involved, such as the distance of the electrode from the endocardium, the curvature of the septum and consequent tangential effects of wave fronts, and others still unknown, make the precise origin, on the basis of these experiments, uncertain.

The time of the R peak was early (0.004 to

0.024 second before the beginning of QRS in Lead I). It was usually simultaneous with an early R in Lead aV_R; an R or a notch on its ascending limb in Lead V_1 or Lead V_2; a Q wave in Lead I when present; a Q wave in leads from the left side of the precordium; an initial positive deflection recorded in the right atrium and in the pulmonary artery or its right branch played one or more distinct notches on its descending or ascending limbs. The latter has already been mentioned as being simultaneous with an r′ in a lead from the pulmonary artery (Fig. 6, V_{RPA3} and V_{RV4}). A distinct notch or slur on the descending limb was often found. This was usually simultaneous with the peak of the R wave in leads from the left side of the

TABLE 3.—*Patient JRo. Time in seconds of QRS deflections in all recorded internal and external electrocardiographic leads measured with respect to the beginning of QRS in Lead I. For locations of electrode when making the intracardiovascular records see teleroentgenogram labelled JRo in Fig. 2. Deflections of simultaneous occurrence and of probable identical origin are indicated by single or double underlining.*

Lead	Q	Notch Ascend. R	R	Notch Descend. S	S	Notch Ascend. S	R′	S′
I	.010		.035		.060			
II			.035		.058		.078	
III			.003		.025		.035	.050
aV_R			.010		.035		.060	
aV_L	.010		.035					
aV_F		.010	.035		.060			
V_1		.010	.025		.035	.060		
V_2			.019	.035	.051			
V_3			.021		.051			
V_4	.010		.035		.060			
V_5	.010		.035		.060			
V_6	.010		.035		.060			
V_{4R}			.010		.035		.060	
Low RPA			.010		.035	.060		
High RPA			.010		.035	.060		
Conus RV			.010		.035	.060		
Outflow RV		.010	.020		.035	.060		
Inflow RV			.010		.035			
Tricusp. RV			.010	.028	.041			
Low RA			.010		.035		.060	
Middle RA			.010		.035		.060	
High RA			.010		.035		.060	
Sup. V. Cava			.010		.035		.060	

(Table 3). The tacit assumption is made that all of these deflections had the same origin, namely early excitation of the interventricular septum from left to right.*

The S wave varied in depth and usually disprecordium (Fig. 4, V_{RV4} and V_4), although not invariably so. For this reason this notch along with the descending portion of the S wave preceding it, was believed to be created by events occurring in the free wall of the left ventricle.

The peak of the S wave varied in time in two principal ways. Sometimes it was simultaneous with the peak of the R wave in left-sided pre-

* Simultaneity of deflections may be fortuitous. But if, in addition, they have a direction in multiple leads ascribable to a single spatial electromotive force, the likelihood of identical origin is very great.

cordial leads (Table 3 and Fig. 6). In such instances there was usually a notch or slur on the ascending limb which had a time similar to the nadir of the S wave in the left precordial leads and the late R or R′ in lead aV_R. In others the nadir of S was simultaneous with the S wave in leads from the left side of the precordium (Fig. 4, V_{RV4} and V_4). These findings support the contention that the S wave in the right ventricular cavity is made up of several components, and its deepest portion has at least two different origins in different subjects.

A late large R followed by an S wave was encountered in the upper ventricle of two patients (PAq and JPHe). In both of these it was preceded by a Q (Fig. 5, V_{RV6}). In a third patient (APy) there was a large R′ preceded by an rs (Fig. 4). In this patient the R′ varied in size from moment to moment presumably caused by movement of the electrode.

With two exceptions the T wave was negative in the cavity. The two exceptions again were patients APy and JPHe. The former showed a large R′ and positive T wave at the basal region, although an inverted T wave was encountered at the apex (Fig. 4). The latter, in whom exploration was quite detailed, showed a positive T wave in all parts of the right ventricle except in the region of the tricuspid valve. This patient showed anomalous atrioventricular excitation intermittently and perhaps cannot be regarded as normal in the electrocardiographic sense.

Since all of the patients with a negative T wave in the right ventricle displayed a positive T wave in the lead from a precordial point over the intraventricular electrode the conclusion would seem reasonable, though not absolute, that a gradient usually exists across the free wall of the right ventricle of such orientation as to suggest that repolarization takes longer on the endocardial than on the epicardial surface.

Premature Systoles. As expected, premature systoles, both of atrial and of ventricular origin, were more frequent when recordings were made while the electrode was being moved in the heart. Undoubtedly they were caused by mechanical stimulation of the walls by the catheter but it could not be assumed that the tip of the instrument was always the stimulating agent. It is true that ventricular premature systoles were more frequent when the electrode was in the ventricle, and atrial premature systoles more frequent when it was in the atrium. However, the reverse occurred, and of the two possibilities it was more usual to encounter atrial premature systoles while the electrode was still in the ventricle. Only once did the opposite set of events occur (patient JNe).

The form of QRS of the ventricular extrasystoles was predominantly negative but this varied; the accession deflections of the premature atrial systoles were always negative.

There did not seem to be any particular region in which extrasystoles were more frequent since they were seen with the electrode anywhere. There was the impression that they might be a little easier to produce in the conus just below the pulmonic valve but this might have been more apparent than real. With the electrode in the pulmonary artery the catheter makes a rather sharp curve backward, to the left, and then to the right. As it is withdrawn into the conus the uncoiling process probably results in considerable pressure either on the free wall of the conus or on the adjacent interventricular septum. The impression may be based on this variable.

Ectopic Rhythms. These were seen in 3 patients as the electrode was moved in the outflow tract. In two instances a rhythm indistinguishable from anomalous atrioventricular excitation (Wolff-Parkinson-White syndrome)[25] developed, once while the electrode was still in the pulmonary artery, and once while being withdrawn through the right ventricle. These phenomena, which reawaken interest in the "double rhythm" theory of the syndrome,[29] have been reported elsewhere.[30]

In one instance runs of ventricular premature systoles with coupling appeared after moving the electrode within the right ventricle.

Demarcation or Injury Potentials. Elevation of the trace to a new level immediately after the inscription of the deflections resulting from depolarization was observed frequently in the ventricles. This displacement was always positive in intracardiac leads, a fact ascribed by

current theory to a subnormal degree of depolarization at the site of pressure by the electrode. However, the pressure could be such as to change the direction only of the normally inverted T wave and to prolong slightly the duration of electrical systole. In such instances it is probable that pressure and subsequent incomplete depolarization was sufficient to balance or slightly overbalance the normal gradient in repolarization presumed to exist from endocardial to epicardial surfaces. Further, the more marked S-T elevations were accompanied by alterations in QRS occasionally of such extreme nature as to make the curve monophasic (Fig. 7) as described by others.

The *P wave* in the right ventricle was usually a low positive deflection. At the base, both in the pulmonic and tricuspid regions, a diphasic nature could rarely be made out (one instance in each region). As expected the atrial regression deflections were poorly recorded from within the right ventricle.

Records from within the Right Atrium

Records were made from the right atrium in 12 of the 14 subjects. With regard to the gross features of these records there seems to be fairly good agreement in available reports.[2, 3, 7, 9, 10] The initial rapid or accession deflections are most often diphasic in character. There is a tendency for the initial positive wave to be larger as the electrode is placed farther from the sinoatrial node. However, with regard to the details of these initial rapid deflections and the presence or absence of others, there is some difference of opinion. Further, the true nature of the regression deflections in the human atrium seems to have escaped most students of the subject.

Atrial Accession Deflections. At the outset it must be kept in mind that the electrode in the atrium, as in the ventricle, may be at a variable distance from the endocardium with a variable amount of blood intervening.* Further it undoubtedly is shifted from moment to moment by systole of the chamber and by movement of the blood. Both of these possible variables must be taken into account when making interpretations.

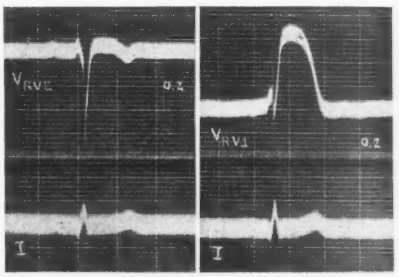

Fig. 7.—Patient JCo. Leads from adjacent points in the right ventricle showing the "monophasic" distortion which occurs (V_{RV1}) when the electrode makes pressure on the endocardium. The similarity of the earliest parts of QRS in both records is to be noted. The simultaneous lead is I, recorded at somewhat less than normal sensitivity with V_{RV2}. Time lines, 0.2 sec.

The nomenclature available for atrial deflections is multiple and unsatisfactory. Lewis[23] designated the initial deflections as extrinsic

* In general the interval between the maximum and minimum peaks of the diphasic accession deflections is greater than the actual interval which separates the source and sink of the doublet responsible for them. This difference is greater the longer the distance between the electrode and the muscle.[26] It has been demonstrated analytically and experimentally by McLeod[22] that when the distance which separates the elements of the dipole is equal to or greater than the length of the muscle being studied it is accurately measured by the interval between the maximum and minimum peaks of the electrogram. In the only measurement of a similar kind made in an abnormal atrium of man[31] on the assumption that the rate of atrial conduction was 2000 mm. per sec. the length of the train of doublets of accession was calculated as 10 mm. and of the doublets of regression as 150 mm. If the usual figure of 1000 mm. per sec. is accepted as the rate of atrial conduction (and our later experiments to be reported favor this) these figures become 5 mm. and 75 mm. respectively. Since the distance of the electrode from the endocardium was unknown it is probable that either figure for the duration of increasing activity is too high, and that errors may be expected in the determination of this interval from the distance between the two ends of the intrinsic deflection thus recorded. On the other hand the duration of the regression process, when measurable, is probably independent of the distance of the electrode from the endocardium because of its length.

and intrinsic depending on whether they were caused by electrical events distant from or immediately under the electrode in direct leads from the dog's atrium. Although the term extrinsic was used to describe a positive deflection preceding the intrinsic one, it is possible to think of extrinsic effects on an exploring electrode of an electrically negative nature occurring after the intrinsic deflection. Hering[32] designated the deflection resulting from the atrial recovery process as the Ta wave. Hurst Brown[24] on the basis of studies on esophageal leads recognized Sn, a, e, i, and o waves resulting from the atrial excitatory process. The Sn and a waves were negative deflections. The first, ascribed to sinoatrial activation, occurred quite consistently almost 0.018 second before the onset of the P wave in Lead II; the second, really a continuation of Sn but occurring after the onset of P_2, was said to represent "the summation of activation effects of parts of the auricle distant from the electrode." The e was the positive deflection comparable to the extrinsic deflection, and i was the rapid change from positive to negative called by Lewis the intrinsic deflection. The deflection o was the gradual return of the trace toward the baseline after the end of the intrinsic deflection. No mention was made by Hurst Brown as to the possible partial origin of this deflection from events during recovery. Battro and Bidoggia[3] designated the terminal slow portion of this o deflection u and believed it to be caused by repolarization effects. In a masterful analysis of the nature of the T wave Macleod[22] was able to show in direct leads from a strip of frog's auricle two peaks, one positive and one negative, during the depolarization, or accession process as it was called, which he labelled a_1 and a_2. During the repolarization or regression process there were also two gradual peaks with reversed polarity which he labelled r_1 and r_2. Hecht[2] has suggested that the atrial deflections all be indicated by P with a subscript to indicate the similarity of the wave to ventricular deflections. Hence there would be a P_Q, P_R, P_S, and P_T. Levine and his associates[9] have expanded Hering's terminology to the atrial accession deflections speaking of a Q_a, R_a, and S_a.

There are advantages and disadvantages of each type of terminology. In the present study it has been possible to identify the intrinsic deflections from each atrium in the record made from the right atrium only, which complicates the matter further. The nomenclatures of Lewis, Hurst Brown, and Macleod are all based more or less on the supposed physiologic origin of the deflections, whereas Hering's and Hecht's are arbitrary as were Einthoven's original designations for the ventricular deflections. The latter approach is perhaps the most desirable at a time when the physiologic origin of all the deflections is still not entirely certain. Perhaps the method of Hering expanded by Levine and his group has a little advantage over Hecht's method, although the choice is largely one of personal preference.

An initial positive deflection preceding the P wave, such as was obtained by Hecht and called a "pre-auricular deflection" because it occurred 0.05 second before the P wave in a simultaneously recorded Lead I or precordial lead, was not observed in the present series. However, both of Hecht's patients had disease of the heart.

An initial negative deflection (Q_a, not QS_a) was encountered in 3 subjects. In two it was found in leads from the upper part of the atrium; in one it was found in leads from upper and lower levels but was inconstant. It occurred between 0.002 and 0.012 sec. before the simultaneously recorded P wave in Lead I. The concept, based on measurements in the dog,[23, 33] is that this early negative deflection is caused by activation of the sinoatrial node itself. It does not seem likely that this structure is capable of giving rise to an electromotive force which can be recorded by the string galvanometer. A. Q_a occurred in at least 1 subject almost simultaneously with the beginning of P in Lead I. Further, that some part of it occurs before the P wave in a bipolar lead does not necessarily favor its sinoatrial origin because the beginning of the latter may be quite late for obvious reasons. Assuming it is analogous to the S_n of Hurst Brown, the constancy of its timing (0.018 sec. before P in Lead II) in esophageal leads is a more cogent argument in favor of its sinoatrial origin.

From the few observations we have made it would appear that the electrode is near the sinoatrial node when this deflection appears but the mechanism of its creation may be simply a matter of orientation of the electrode.

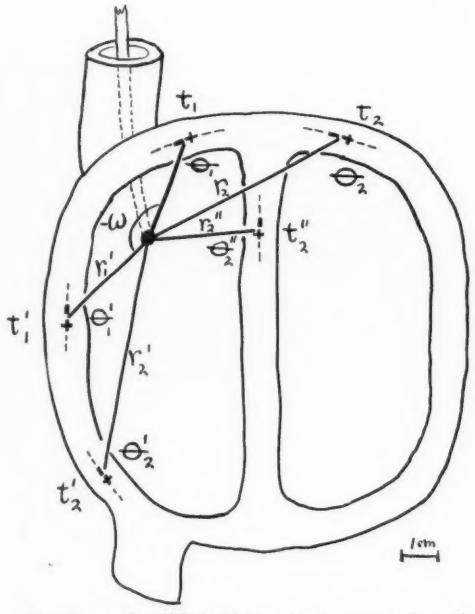

FIG. 8.—A simplified, schematic representation of a frontal section through the atria just anterior to the venae cavae to illustrate how the intra-atrial P wave may begin with a negative deflection (solid angle—ω), and later be characterized by a positive deflection as a dipole approaches the electrode through the septum (t_2''). The effects of dipoles at t_2 and t_2', being relatively distant (r_2 and r_2') are extrinsic and negative. The diagram serves to illustrate the cause of such bizarre P waves as seen in complexes 10 and 11 of figure 9. Not illustrated are the effects of the regression process. In making the diagram, a rate of conduction of 1000 mm per sec. was assumed.

The times t_1, $t_1' = 0.002$ sec. and t_2, t_2', $t_2'' = 0.006$ sec. from the beginning of the atrial excitation. Potentials of the electrode are determined by the equations $V = \mu \cos \theta / r^2$ and $V = \phi\omega$.

It is visualized that at first the electrode is dominantly affected by the active side of an umbrella-like wave of excitation preceding in all directions from it (Fig. 8, solid angle subtended at the electrode at times t_1 and t_1'). Later the electrode is affected by an oppositely oriented wave front on another side (septal) of the atrium (t_2''). The figure is exaggerated but it is conceivable that something of a similar nature may account for a Q_a which precedes the beginning of the P wave in the standard leads.

The figure also helps to explain later intrinsic-like deflections probably arising in the left atrium occasionally seen when the electrode is near the mouth of the superior vena cava and rarely in other locations (Fig. 9, complexes 10 and 11). In one instance during the

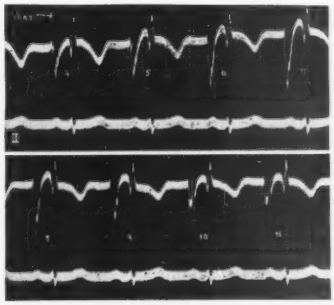

FIG. 9.—Patient SAt. A continuous record made simultaneously with Lead II as an exploring electrode was drawn slowly upward through the right atrium beginning at point 9 (Fig. 2). The change in Ra follows no fixed pattern but Sa at first gets deeper, then shallower, and in complexes 10 and 11 a late intrinsic deflection is superimposed on its ascending limb. The slurred ascending limb of S in earlier P waves is in part an accession effect and in part a regression effect. The gradual elevation of the P-R segment is ascribed to the positive phase of regression which is expected as the upper end of the atrium is approached. The large R of QRS is to be noted.

transition from the right atrium to the superior vena cava a slow slurred initial depression occurred 0.022 second before P_1. This was considerably earlier than in any other complex of the strip and may have been due to sinoatrial activation.

Although in general the e wave (R_a) was taller in leads from lower than in leads from higher levels this was not invariably the case. There are several reasons why this might be so, possibly the most important being the distance of the electrode from the endocardium in each instance and the relative orientation of the electrode with respect to extrinsic effects which as noted may be negative as well as positive.

The o wave (ascending limb of S_a or QS_a) is most prominent in those leads which show a small or absent extrinsic deflection. It is broad and slurred and is probably in large part caused by excitation of atrial muscle in a direction away from the electrode. This statement is based on the fact that when the electrode is placed near the left auricle (appendage) in the pulmonary artery, the comparable deflection is sharp and rapid, not slurred (Fig. 3, V_{PA4}). Further, its termination is simultaneous with the termination of the o in the lead from the region of the sinoatrial node in the right atrium (Fig. 3, V_{RA10}). Although these data support its designation as an accession event, nevertheless, there are certainly included in it the earliest negative portions of the regression process. This point can be seen in Figure 9 where the dipolar nature of recovery can be made out in part by progressive elevation of the P-R segment as the intrinsic deflection becomes deeper (complexes 5 to 8).

Atrial Regression Deflections. In the normal subject the recognition of either r_1 or r_2 is difficult. The former is obscured by o, the latter by the advent of ventricular excitation. Nevertheless, in records made from near the sinoatrial node, the P-R segment is usually slightly above the baseline (Fig. 9), and in records made distant from this site the reverse displacement of the segment occurs, an observation made by Battro and Bidoggia.[3] This fits in well with what Macleod[22] has predicted by regarding the regression process as essentially dipolar in nature. Recognition of the initial part of this diphasic deflection is occasionally possible in human records (Fig. 9), especially if there is some degree of atrioventricular block. Further, there is a fifth deflection in these abnormal records which is late, positive, and gradual and is comparable in every way to the U wave encountered at times in records of the ventricular action potential.

Duration and Rate of Conduction of Action Currents in the Atrium. Measurements have been made on the duration of the excited state in the atria of animals[22] and of man.[31] In a group of normal human subjects such measurements cannot be made easily because the terminal portions of atrial regression are obscured by the deflections resulting from ventricular excitation. Considerable error may be made in the measurements, especially on the duration of the accession process, if the electrode is at a distance from the endocardium (see * page 21). Nevertheless an attempt was made to approximate the duration of accession by measuring the time between the beginning and end of the intrinsic deflection in representative records made from different levels of the atrium. The maximum, minimum and mean values obtained were 0.003, 0.025, and 0.010 sec. respectively. It is probable that the minimum value is the closest to the true value.

On several occasions, both in normal subjects and in patients, a calculation of the rate of conduction in the atrium was attempted. This was done by measuring the distance on the chest wall between the frontal fluoroscopic projections of the electrode at two different points in the right atrium. The difference in time between the beginning of the intrinsic deflection in leads from these two points was then taken as the time consumed as excitation traveled over the measured distance. In retrospect such measurements are extremely crude, particularly the measurements of the distance, and involve certain assumptions which in the light of our later experience with a double electrode catheter are not justified. Until the newer technics have been explored further it is probably best to assume that the rate of conduction in the human atrium is similar to that in the dog,[14] namely 1000 mm. per second.

The mean difference between the peaks of R_a in upper atrial records and in lower atrial records was 0.032 sec.; between the nadirs of S_a waves in the same records it was 0.033 sec. This crude measurement means that excitation reaches the lower levels of the right atrium about 0.033 sec. later on the average than it reaches the upper levels.

Demarcation Potentials in the Atrium. Presumably from pressure of the electrode on the atrial muscle a positive displacement of the baseline before the completion of the intrinsic deflection occurred on one occasion (Fig. 10). This is suspected of being caused by an injury potential or demarcation potential[27, 28] developed as a result of block, either partial or

complete, of the depolarization process near the pressure-making electrode such as is seen in direct leads,[34] and as was seen in the ventricle (Fig. 7).

It was immediately felt that some practical use could be made of this phenomenon to make an electrographic diagnosis of atrial septal defect. A record with a late intrinsic deflection and elevated P-R segment would be expected if

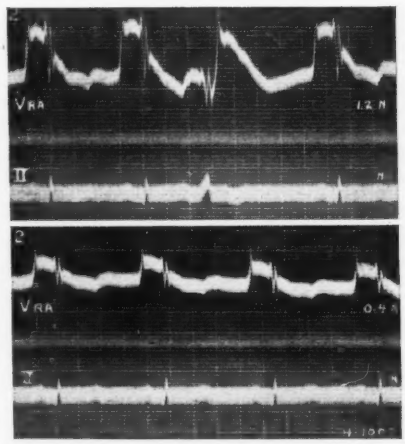

FIG. 10.—Patient JNe. A lead from the right atrium recorded simultaneously with Lead II showing the effects on the P wave of pressure of the electrode in the endocardium. The Sa wave is absent and the P-R segment is elevated. The lower row is the same as the upper except for the increased tension of the string (1.2 N to 0.4 N). In the upper row the fortuitous occurrence of a ventricular premature systole permitted a good record of the total duration of atrial systole (approximately 0.04 sec.) in the following cycle. The quadriphasic nature of the ventricular complex is to be noted. Time lines, 0.2 sec.

pressure was made with the electrode on the endocardial surface of the left atrium, and indeed such a record has been obtained in a patient with an atrial septal defect. However, a similar one has been encountered in a patient with a normal sized heart and suspected pulmonary arterial hypertension. Further, such electrograms have been encountered in normal subjects by Levine and his associates[10] although these investigators felt that an orifice of some kind existed in the interatrial septum and that the electrode was in the left atrium. In the light of our experience and of more recent findings[35] the electrode was undoubtedly in the coronary sinus or one of its venous branches. Under such circumstances the intrinsic deflection is later, by virtue of the electrode's proximity to the left atrium. Further, sufficient pressure clearly may be exerted through the sinus wall to cause impairment of depolarization of atrial muscle and consequent displacement upward of the P-R segment. The finding, therefore, cannot be regarded as diagnostic of persistent atrial septal defect.

Ventricular Deflections Recorded from the Atrium. In the 12 subjects with records available from the atrium the QRS began with a positive deflection in seven, with a negative deflection in four, and in one with either a positive deflection or a negative deflection depending upon the location of the electrode in the atrium.

The initial positive deflection, when present, was always simultaneous with a similar wave observed in leads from the tricuspid region of the right ventricle, the superior vena cava, the right branch of the pulmonary artery, and the right arm. An initial Q, when present, was usually simultaneous with the nadir of a similar deflection in the leads from the last three locations just mentioned, and with the nadir of S or the notch on its descending limb in the lead from the cavity of the right ventricle. A late R′ might be small in all atrial locations and simultaneous with a similar deflection in records from above the atrium including the right arm, or with a notch on the ascending limb of S in the ventricular electrogram from the tricuspid region. On two occasions (patient QWi and patient SAt, Fig. 9) this deflection became quite large as lower atrial levels were approached, only to disappear as soon as the electrode was placed in the ventricle. In one of these (SAt) it was also large in the pulmonary artery. In two other instances (patients APy and PAq, Figs. 4 and 5) already mentioned, this late R was still prominent as a large positive deflection in a lead from the tricuspid region of the right ventricle, and could also be identified in the lead from the pulmonary artery.

In general the records obtained in the upper part of the right atrium were simulated quite

faithfully by leads from the right arm, but this relationship was not close in the case of leads from the lower atrial levels.[2]

In *summarizing* what was found in the atria it may be stated that to begin with the electrical processes in atrial muscle consist of two parts, excitation and recovery, and that each of these, from experimental data, are dipolar in nature. An electrode in the atrium may be affected by both of these processes from one portion of the chamber and at a slightly later time by one or both in distant parts of the chamber. This overlap makes a precise separation of the resultant deflections difficult. It is suspected that initial negative deflections are the result of the electrode being near the sinoatrial node and on the active side of myocardium from the very beginning of atrial excitation. It cannot be stated with certainty from the data presented that activation of the sinoatrial node itself causes any part of initial negativity of the right atrial cavity, although this is possible.

With regard to ventricular deflections, an initial positive wave was usually simultaneous with other deflections ascribable to early depolarization of the left side of the interventricular septum. A large negative deflection, regardless of its location, was usually simultaneous with the peak of the R wave in leads obtained from the left side of the precordium and with the S wave or notch on its descending limb in the right ventricular lead suggesting its origin as being from excitation of the free wall of the left ventricle. The late positive deflection of large size in four subjects, and of moderate size in five others, was ascribable to some considerable mass of muscle excited from below upward and from left to right to be discussed below.

Records from Within the Superior Vena Cava and Other Veins of the Thorax

Records were made from the superior vena cava in nine subjects. In all the P wave was inverted, and usually bore several notches. The QRS in five consisted of the rS type, in three of which a distinct notch was present on the ascending limb of S. In two QRS had the form of Qr, in one of qR, and in one of rSr′. The T wave in all was inverted. The similarity to the lead from the right arm was striking in all except for the lower amplitude and the occasional slurring rather than notching of the ascending limb of S in the latter. In only two was a simultaneous record made from the precordium with an electrode directly over the intravenous one. In both of these (patients JRo, Fig. 6, V_{SVC10}, and SAt) the P and T waves were similar in the internal and external leads but the QRS was different, in one instance markedly so.

Toward the end of each experiment the electrode was placed fluoroscopically well out to the left border of the thorax (Fig. 2), usually, as far as could be judged, in the left axillary vein. In general, records from this region were similar to what was obtained in leading from the left arm although there were a few exceptions. Other records between the superior vena cava and this point were often attempted but were usually technically difficult because of large, irregular electromotive forces presumably of venous origin.

The Large Positive Deflection at the Base of the Right Ventricle

What is believed to be one of the most significant observations in this study was the large positive deflection found in the lower right atrium of four subjects and a similar deflection of moderate size in several others (Figs. 4, 5, 6 and Table 3). The simultaneity of this wave with a variety of others in leads from within the heart and from the surface of the body have been alluded to earlier. In brief it appears that the electromotive force responsible for the deflection is large enough to be reflected in leads from the surface of the body and this force, regarded as a vector, seems to be, at least in the cases studied, pointing to the right and upward. Whether its direction is backward or forward appears to depend in part on the position of the heart in the thorax and undoubtedly on such other factors as the relative size of the ventricles, their exact orientation with respect to each other, the orientation of the muscle responsible for the deflection with respect to various points on the surface, and

possibly the architecture of the Purkinje system. When the force is pointing well forward, it is the cause of the r′ in the rSr′ complex sometimes encountered in normal subjects in a lead from the right sternal edge or from points farther to the right on the thorax. We have had the opportunity of confirming this in one subject not included in the present series. Also, when directed forward, it may cause a negative notch on the ascending limb of the S wave in the lead from the superior vena cava (Fig. 4, V_{SVC5}).

In the past this deflection or something similar to it has been ascribed, largely from the results of direct leads in dogs,[36, 37] to late excitation of the "conus." No statement has been made as to the particular part of the conus concerned although it has been generally assumed that it was the free wall. It has also been stated that a late positive deflection (r′) may be found frequently in precordial leads taken over the conus.[38]

There is much evidence which tends to refute both of these concepts. With regard to the dog it is significant that records may be obtained from the free wall of the conus which have a configuration, rsR′s′[37, Fig. 3E] not unlike what was seen in an internal lead from the conus in patient APy (Fig. 4, V_{RV1} and V_{RV3}). The possibility exists that the peak of r really represented the early arrival of excitation at the epicardial surface of the conus and that R′ was transmitted from deeper-lying muscle which was excited quite late. We have made no observations in animals but the point is worth investigating.

With regard to man, in the only direct lead made from the conus[39] it was almost the earliest surface point to be excited (0.0143 sec. after the beginning of R in Lead II), and at all ventricular points, including many on the posterior surface of the left ventricle, the intrinsic deflection began before half of the QRS interval was completed. The deflection we have observed had a time usually well beyond the midpoint of the total ventricular depolarization interval. Further, it was positive in the lower atrium and in the pulmonary artery, and negative in leads from the precordium near the conus (Figs. 5 and 6), both almost impossible to explain on the basis of late excitation of the free wall of the conus either radially outward, or longitudinally toward the pulmonic valve. Lastly, the free wall of the conus is a relatively thin structure, and when it differs quite markedly in a physiologic way from the cardiac muscle, or is particularly devoid of Purkinje fibers, it should not be excited very late.

The particular muscle mass which gives rise to the deflection under consideration must be, in the light of our present knowledge, conjectured. A specific observation regarded as important in localizing it is that the deflection, with two exceptions, underwent an abrupt change from a summit to a depression as the electrode slipped through the pulmonic valve on its way to the right ventricle. A similar but reverse behavior of the record was seen as the electrode traversed the tricuspid valve on its way from the right ventricle to the right atrium (Fig. 4). These data suggest that the particular muscle concerned was common to both orifices. Lastly, the structure must be at the base of the right ventricle, a statement supported by what has already been presented and also by the observation that the late deflection, when encountered in the ventricular cavity, was at the base of that chamber.

An anatomical structure which meets these requirements is the crista supraventricularis[40] in the roof of the right ventricle. It is a thick muscular ridge, 12 to 15 mm. high, which extends forward and to the left from the atrioventricular to the pulmonic orifice.[41] The ridge divides the upper part of the ventricular cavity into an inflow tract and an outflow tract, the latter being the conic interior of the right ventricle recognized on the surface as the conus arteriosus. At the anterior septal end of the ridge the origin of the moderator band (trabecula marginalis) may be found.

If this ridge is adequately supplied by subendocardial Purkinje fibers its anatomical position favors its mean direction of excitation upward, and to the right, a conclusion reached earlier from the direction of various deflections presumed to have their origin in this particular muscle band.

This crista supraventricularis undergoes con-

siderable hypertrophy in diseases causing increased work of the right ventricle, and it is suspected that the electromotive force to which it gives rise normally may be the same one responsible for some of the late positive deflections found in various external leads, especially on the right side of the precordium, in right ventricular hypertrophy. A preliminary survey of our observations on patients with right ventricular hypertrophy suggests that this is so, but a precise statement will depend upon a more detailed analysis of our records than we have thus far been able to make. Whatever the area concerned, it is by no means the last to be excited in the ventricles, for the deflection or deflections resulting from its excitation occur during the third quarter of the QRS interval.

The matter has importance because of the considerable bearing it has on several fundamental electrocardiographic problems. One of these is the problem of the origin of the late R wave or R′ in leads from the right side of the precordium in right ventricular hypertrophy and in right bundle branch block already mentioned. Another is the applicability of a vector type of recording of the human electrocardiogram as a possible means of reducing the number of leads that need be taken for clinical purposes. The observations presented suggest that the reduction of all the ventricular electromotive forces to a single vector is probably too gross for practical value. However, it would appear that there are three principal vectors, so far as QRS is concerned, which would give practically all the information desired at least in the normal heart, and possibly in the abnormal heart too. These vectors, briefly, are the three responsible for the "Q", "R", and "S" respectively. In terms of what is known and what has been presented, the "Q vector" is produced by early excitation of the left side of the septum; the "R vector" by excitation principally of the apical free wall of the left ventricle; and the "S vector" by excitation probably of muscle in the roof of the right ventricle in a direction which is variable but dominantly upward, backward and to the right.

Studies are under way in which vectorcardiograms and surface null potentials of the three principal vectors are being recorded in an effort to delineate their precise orientation in the thoracic space, and to test their usefulness in clinical diagnosis.

Relations between the QRS of Intracardiac and of Precordial Leads

Of particular interest in the normal precordial electrocardiogram has been the origin of the deflections in leads especially from the right side.

From what has gone before it is clear that in Leads V_1 and V_2 both the R wave and the S wave have dual origins. In high-speed records the R wave is usually notched either on its ascending or descending limbs. Either the notch on the ascending limb or the peak may be simultaneous with the peak of the R wave encountered in the lower portions of the right ventricle. In the former instance the peak, and in the latter instance a notch on its descending limb, occur between 0.01 and 0.02 second later. It is suspected that the initial part of the summit in Lead V_1 is septal in origin, and its later part is ascribable to excitation probably of the free wall of the right ventricle. This point has been made previously.[5]

The S wave in Leads V_1 and V_2 may show a notch on the descending limb simultaneous with the peak of the R wave in leads from the left side of the precordium. Under such circumstances the nadir is simultaneous with the nadir of S in Leads V_4 or V_5 or occurs at a time which is somewhere between the time of R and S in the left-sided leads. Although it occurs, it is exceptional for the nadir of S in leads V_1 and V_2 to be simultaneous with the peak of the R wave in Leads V_4 and V_5. It appears, therefore, that in the normal subject the S wave, both in leads from the right and left sides of the precordium, is produced largely by the electromotive force which gives rise to the large positive deflection often found in the right atrium and pulmonary artery and occasionally inside of the base of the right ventricle.

As to the leads from the left side of the precordium, the origins of Q and R are fairly well proven. The origin of the S wave has been suggested by observations and discussion given above.

Summary

1. Simultaneous galvanometric records were made of the potentials encountered in the right heart and attached vessels in 14 patients without clinical evidence of heart disease.

2. An analysis of these records and their temporal relationships to semidirect and indirect leads has been presented. The relationship of precordial to intracardiac QRS deflections has been elaborated particularly.

3. The significance of multiple deflections in the P wave recorded in the atrium, and of large positive QRS deflections encountered in proximity to the base of the right ventricle has been discussed.

4. It is suggested that the crista supraventricularis of the right ventricle may be the source of a large, moderately late electromotive force which is reflected not only in intracardiac but also in surface leads.

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Peri-Infarction Block; Electrocardiographic Abnormality Occasionally Resembling Bundle Branch Block and Local Ventricular Block of Other Types

By SAFETY R. FIRST, M.D., ROBERT H. BAYLEY, M.D., AND D. R. BEDFORD, M.D.

Curves which represent bundle branch block can usually be identified. More important are those which represent peri-infarction block. Multiple unipolar leads enable identification of the latter from the former, which they may closely resemble in the limb leads, and from intraventricular block of other types. Criteria and examples of peri-infarction block are presented and an incidence and explanation are offered.

THE IMPORTANCE of the subject of intraventricular block has recently been emphasized by Wilson and associates.[1, 2] In many instances the confusion results from similarity of the electrocardiographic pattern in various types of interventricular block. In most instances complete right and complete left bundle branch block can be properly identified. On the contrary, incomplete grades of these lesions are apt to be confused with marked hypertrophy alone or in combination with bundle block.

Equally as distinctive as complete bundle branch block is the lesion we shall call *peri-infarction block*.* In the vast majority of instances it can be differentiated from bundle branch block and from intraventricular block of other types. The importance of the lesion is emphasized, as the name implies, by its association with (usually) an old myocardial infarction. Wilson and his associates have preferred to refer to the lesion as "arborization block," indicating that they do not necessarily agree with the implications of the name "arborization block."[1, 2]

It is the purpose of this communication to describe the essential features of the electrographic pattern of peri-infarction block and to present electrocardiograms which illustrate the lesion as produced by myocardial infarctions of the common locations.

The diagnostic criteria of peri-infarction block include: (1) evidence of myocardial infarction of the subendocardial region of the ventricular wall, or transmural infarction with a circumferential region of subendocardial damage; (2) QRS complexes in the limb leads which are, or exceed, 0.11 second in duration; (3) a characteristic QRS pattern in the unipolar precordial and extremity leads.

When the exploring electrode is placed over the center of a transmural infarction with a circumferential region of subendocardial damage, the associated deflection will be of a QS type represented in figure 1, *a*. As the exploring electrode is moved over the living muscle comprising the outer layer of the involved part of the ventricular wall, there is late activation of this region and the electrocardiogram (figure 1, *b*) demonstrates an initial Q wave followed by a broad R wave, the downstroke (intrinsicoid deflection) of which terminates the long QRS interval. It follows that, when the exploring electrode is placed directly over an infarction which involves only the subendocardial lamina, tracings of the latter type, namely, a Q wave with a broad R wave, will be obtained. In electrocardiograms recorded from the nor-

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* This term was suggested by W. T. McCollum, M.D., Department of Internal Medicine, University of Oklahoma School of Medicine, Oklahoma City, Okla.

mal ventricular wall adjacent to the infarction the late activation of the blocked region in a direction away from the exploring electrode produces a negative potential equal to the positive potential produced by late activation in a direction toward the electrode. The intrinsicoid downstroke of the R wave is relatively early and the remainder of the QRS interval is written on the base line (fig. 1, *c*). Electrode positions across the chamber from a subendocardial infarction (fig. 1, *d*) yield QRS complexes char-

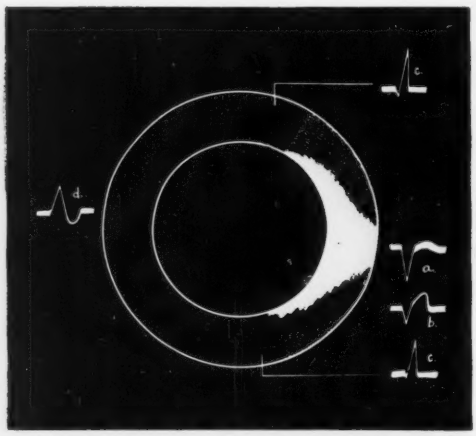

FIG. 1.—Diagrammatic illustration of the various forms of the QRS interval encountered in peri-infarction block. The whitened area indicates infarction while the stipled area indicates a circumferential area of blocked normal muscle tissue overlying the infarction.

acterized by an R wave which is followed by a broad S wave. The broad S wave indicates late activation of the blocked, living muscle tissue overlying the infarcted region. The magnitude of the R wave will depend primarily upon contributions from the uninvolved wall subjacent to the exploring electrode.

METHODS AND CASE MATERIAL

In addition to the standard and unipolar limb leads, multiple unipolar chest leads were recorded by employing a central terminal.[3] In some instances it was necessary to explore regions higher and lower than the six standard precordial positions.[4] In such cases, subscripts (NH) or (NL) have been used to indicate the regions from which tracings were obtained, where N denotes the number of interspaces deviated from the normal and H and L the direction high or low. For example, leads recorded two interspaces higher, and on the vertical with the standard V_6 lead were labeled $V_{6(2H)}$.

The electrocardiogram, figure 2, was recorded from a white woman, 65 years of age, whose history

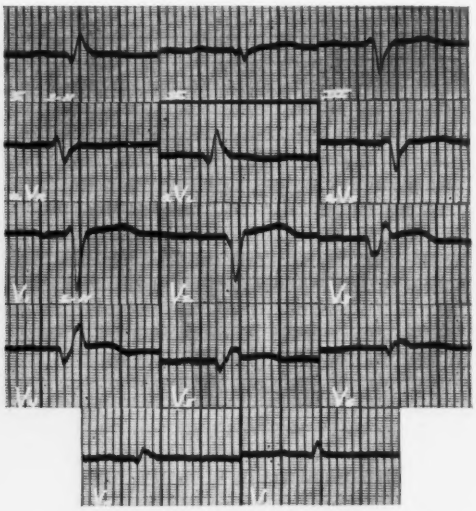

FIG. 2.—Anterolateral and lateral infarction of the subendocardial portion of the myocardium with peri-infarction block. The tracings on the limb leads closely resemble those seen in bundle branch block, while the precordial leads demonstrate normal-appearing deflections in V_1 and V_2. Deflections V_3 through V_7 are of blocked form and indicate the block is local, which would not be the case if one of the His bundles were involved. The form of the RS-T segment suggests that infarction might be moderately recent. The history indicated that infarction occurred three years previously and an electrocardiogram at that time, not reproduced, showed anterolateral infarction without peri-infarction block.

indicated myocardial infarction of three years' duration. The QRS interval is 0.14 second. The R wave is absent in Lead V_2, and there is a broad Q wave followed by an R wave with a late downstroke, terminating the QRS interval in V_3 through V_7. Positive RS-T injury shift is present in Leads V_2 through V_6. Lead V_8 has a normal form and the chief downstroke (intrinsicoid deflection), which apparently terminates the QRS interval, occurs 0.08 second earlier than the corresponding downstroke in V_3. These changes are considered characteristic of anterolateral and lateral infarction of the subendocardial portion of the myocardium with peri-infarc-

tion block. The fact that curves of block form with a late intrinsicoid deflection (Lead V_3) are recorded from one region of the precordium and a normal curve, V_5, from an adjacent region on the same side

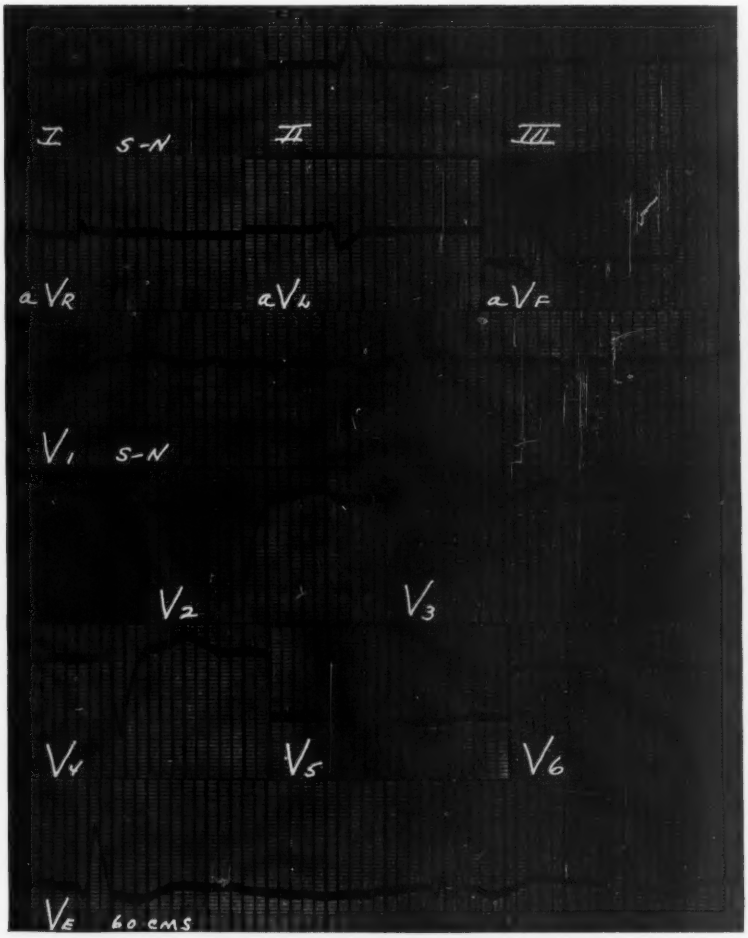

FIG. 3.—Inferoseptal (posterior) infarction with peri-infarction block. The prominent Q wave, and late downstroke of the QRS interval in Leads aV_F and V_E (esophagus, depth 60 cm.) are considered diagnostic in this instance. Auricular fibrillation with occasional ventricular extrasystole is also present.

of transition indicates that the block is local, and not to be ascribed to a lesion of one of the main branches of the His bundle.

Figure 3 is the electrocardiogram recorded from a Negro man, 65 years of age, who, six weeks prior to the examination, experienced severe substernal and precordial pain which radiated to both shoulders and down both arms. Hypodermic injections were required for relief. The QRS interval is 0.14 second and the Q wave is prominent in Leads II and III. The downstroke of the R wave is slurred in Leads aV_F, V_5, V_6, and V_E. There is a prominent Q wave and the intrinsicoid deflection terminates the QRS interval in leads recorded from the left leg and esophagus.

These changes are characteristic of inferoseptal (posterior) infarction with peri-infarction block. Auricular fibrillation is also present and there is an occasional ventricular extrasystole.

The electrocardiogram of figure 4 was recorded from a 70 year old white man ten hours following the onset of symptoms of myocardial infarction. The

QRS interval is 0.16 second. There is a Q wave in all precordial leads which decreases in size as the exploring electrode is moved to the left. The intrinsicoid deflection is late and terminates the QRS complex in Leads V_1, V_2, and aV_F. This deflection is early and is followed by a small S wave in Leads V_5 and V_6. A diagnosis is suggested of moderately recent anteroseptal, anterolateral, and inferoseptal infarction with peri-infarction block, or possibly right bundle branch block, or both.

The absence of an abnormally large S wave over the left side of the precordium (V_5 and V_6) is most unusual in right bundle branch block. Additional leads from positions farther to the right might prove helpful. Double R waves from these positions would indicate right bundle branch block, while an initial R wave with either a normal or prominent S wave, such as appears at the left shoulder, would tend to indicate peri-infarction block. This patient expired before additional studies could be completed.

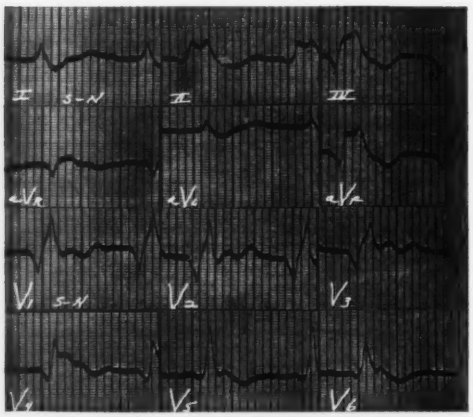

FIG. 4.—Moderately recent anteroseptal, anterolateral, and inferoseptal infarction with peri-infarction block, or possibly right bundle branch block, or both. The limb leads in this instance certainly suggest bundle branch block. The precordial leads, however, fail to demonstrate prominent S waves over the left side of the precordium (V_5 and V_6) which would be expected if the right branch of the His bundle were involved.

The tracing reproduced in figure 5 was recorded from a 46 year old white man six years following a clinical and electrocardiographic diagnosis of myocardial infarction. The QRS complex is 0.11 second. The R wave is small in Lead V_1, preceded by a small Q wave in V_2 and V_3, and is absent in V_4. The S wave is prominent in V_2 and V_3. There is a Q wave with a low notched R wave and late downstroke in Leads V_5 through V_9. $V_{5(2H)}$ reveals a small Q wave and the up-stroke of the R wave is slurred, while aV_L presents an initial R wave, and the down-stroke which terminates the QRS interval is 0.08 second following the onset of the R wave. These changes indicate anteroseptal infarction with a thin, extensive layer of subendocardial damage in the anterolateral, lateral, and posterolateral regions accompanied by peri-infarction block. Incomplete left bundle branch block is also to be considered. The prominent Q wave in leads from the left side of the precordium must then result from initial negativity within the cavity of the right ventricle which could not be transmitted to V_5 through V_9 unless the right side of the interventricular septum were extensively infarcted.

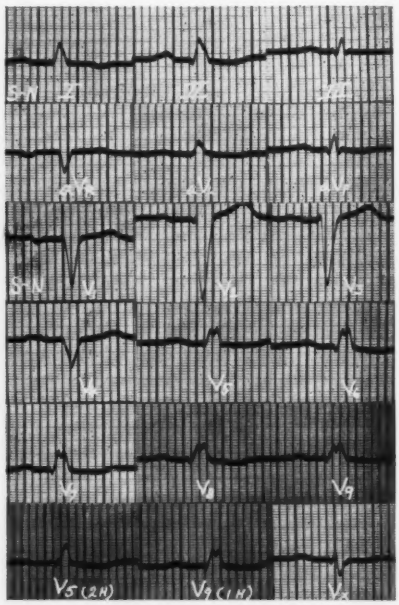

FIG. 5.—Transmural, anteroseptal infarction with a thin, extensive layer of subendocardial damage and peri-infarction block in the anterolateral, lateral, and posterolateral regions. The prominent Q wave with broad, notched R wave and late downstroke in Leads V_5 through V_9 indicate an abnormal order of activation outwards through the free wall of the left ventricle.

The prominent Q wave with a broad, notched R wave and late downstroke in Leads V_5 through V_9 certainly indicates an abnormal order of activation outward through the free wall of the left ventricle. It would be difficult to understand how factors in that region of the ventricular wall opposite to, or across the chamber from, the one under discussion could produce the abnormal R wave in these leads. $V_{5(2H)}$ and aV_L which represent nearly normal left ventricular deflections support the contention that a small subendocardial infarction is present in the free wall of the left ventricle.

Figures 6 and 7 were recorded from a 53 year old physician who had experienced recurrent attacks of angina pectoris. Figure 6 was recorded two days following an unusually severe attack of substernal pain which radiated to both shoulders and was accompanied by profuse perspiration and prostration.

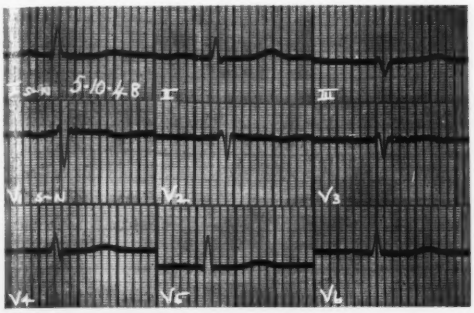

FIG. 6.—Electrocardiogram showing normal tracings; heart in the semihorizontal position. Curve recorded from a 53 year old physician two days following an attack of angina pectoris.

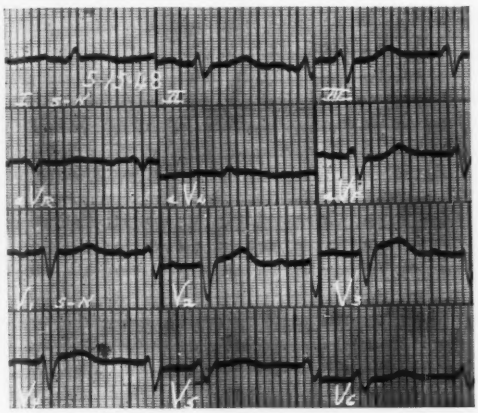

FIG. 7.—Tracing recorded from the same subject and five days following that of figure 6. Recent anteroseptal and high anterolateral infarction with peri-infarction block is now present. aV_L is the only position explored presenting a later downstroke. Additional precordial leads are indicated (see text).

The initial curve was taken when the patient was asymptomatic and there were no positive physical findings. The electrocardiogram shows no abnormality and the heart is in the semihorizontal position.

Figure 7 was recorded from the same subject five days later and six hours following the onset of severe substernal and precordial pain which radiated to the tip of the left scapula and was accompanied by extreme dyspnea and prostration. The QRS complex is 0.11 second and there is a broad Q wave with positive injury shift in Lead I. The R wave is minute in V_2 and V_3, where a positive RS-T junction displacement is also present. V_5 and V_6 demonstrate a small R wave and a broad S wave. Previously, the R wave was not followed by an S wave in these leads. Lead aV_L consists of a broad Q wave followed by an R wave and is the only position explored which presents a late intrinsicoid deflection at the end of the abnormally long QRS interval. These changes suggest recent anteroseptal and high anterolateral infarction with peri-infarction block. The anteroseptal involvement is transmural and consequently uninvolved by the block phenomenon. The patient expired before additional leads from high precordial positions could be made.

DISCUSSION

The exact mechanism responsible for producing a prolonged QRS interval following a subendocardial infarction merits consideration. Wilson and his associates listed several possibilities, chief among which included "a functional depression of the specialized ventricular conducting system or of the ordinary ventricular muscle as a whole, to lesions affecting one or more of the subdivisions of one of the bundle branches, or to involvement of the Purkinje network or subendocardial muscle on the inner aspect of the part of the ventricular wall affected."[2]

Electrocardiograms of 464 subjects demonstrating myocardial infarctions were reviewed in the laboratory of the University of Oklahoma School of Medicine. Unipolar limb and precordial leads were taken in all instances. Of this number seventeen, or nearly 4 per cent, presented the characteristic findings of peri-infarction block. In the limb leads the similarity between subendocardial infarction complicated by peri-infarction block and bundle branch block may lead to an erroneous diagnosis and in most instances differentiation requires multiple unipolar leads. It is our belief that the majority of instances of lesions of the kind presented are interpreted as bundle branch block or intraventricular block of other types.

A permanent peri-infarction block phenomenon usually develops several months after infarction, but occasionally may occur early either in transient or permanent form. We pre-

fer to believe that in subendocardial infarction radial spread from the endocardial surface is usually maintained by conducting strands of living muscle through the infarcted region. Later, as contraction fibrosis occurs, the conducting strands would be subject to pressure atrophy and peri-infarction block would ultimately result. In cases where the block appears early, it would appear that strands of living muscle through the infarct do not exist or do not permit radial spread to the overlying normal wall. Recovery of function of the fibers specified would, however, determine cessation of early temporary peri-infarction block, and failure of recovery would then determine early permanent peri-infarction block. Centripetal spread of activation parallel to the epicardial surface from the circumference of normal muscle overlying the infarct would be the natural result of failure of outward radial spread from the endocardial surface, and since the former takes place at one tenth of the speed normally encountered over the endocardial surface, the associated wave form is a function of the size of the blocked region relative to the position of the exploring electrode.

Summary

1. Peri-infarction block produces a characteristic electrocardiographic pattern which may often closely resemble that of bundle branch block or intraventricular block of other types, particularly in so far as Leads I, II, and III are concerned.

2. Infarctions of the subendocardial region of the myocardium are responsible for the production of peri-infarction block.

3. The term "arborization block" is objectionable since its implication limits the nature of the blocking process by directly incriminating the Purkinje network and, at the same time, fails to emphasize its association with infarction. Moreover, a reasonable explanation appears to depend upon local damage of the myocardium.

4. The limb lead electrocardiogram does not ordinarily furnish sufficient information for the differentiation between subendocardial infarction complicated by peri-infarction block and intraventricular block of other types.

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Excitation of Human Auricular Muscle and the Significance of the Intrinsicoid Deflection of the Auricular Electrocardiogram

By HANS H. HECHT, M.D., AND LOWELL A. WOODBURY, PH.D.

The technic of endocardial electrocardiography has been useful in applying certain basic concepts to the interpretation of an electrocardiogram. One of the earliest intra-auricular records, obtained in 1944, is here analyzed in detail. It led to a reaffirmation of the dipole concept of cardiac excitation. A number of selected auricular electrocardiograms recorded by simultaneous tracings from the chestwall and the esophagus demonstrate the direct clinical usefulness of the theoretic postulates.

ELECTROCARDIOGRAMS of human subjects obtained from the right auricular and ventricular cavities have stimulated re-examination of the dipole approximation of the membrane theory.[1-7] The shape of the electrocardiograms thus obtained has tended to confirm previous concepts which considered cardiac action potentials to arise from a band of dipoles moving over the heart.[8-12] This interpretation is not generally accepted by those who view cardiac excitation and recovery in terms of an "interference theory" which assumes that an electrocardiogram represents the summation of two somewhat asynchronous monophasic action potentials.[13-17] A discussion of suitable direct leads from the auricular surface of the human heart in situ with observations concerning the clinical application of such studies therefore appeared desirable. One of many records obtained from the right auricular cavity was found to be especially suitable for further analysis of the dipole concept.

The record (fig. 1) was obtained from M. S., a 58 year old Greek-American laborer who suffered from arteriosclerotic heart disease with sclerosis of the coronary arteries and right bundle branch block. A catheter containing the electrode was inserted into the right auricular cavity through the right antecubital vein. To avoid injury effects on the electrocardiogram, the tip of the electrode did not coincide with the tip of the catheter (whistle-tip catheter). When an attempt was made to pass the catheter into the right ventricular cavity it was deflected upwards and posteriorly and came to rest against the posteromedial portion of the interauricular septum about halfway between the sinoauricular region and the annulus fibrosus. The location was confirmed by roentgenograms obtained with the patient in the anteroposterior and in the right anterior oblique positions. A Sanborn Tribeam electrocardiograph was used and Lead I was recorded simultaneously with the endocardial records.

The auricular electrocardiogram of figure 1 consists of three parts: (1) a very small deflection, P_p, (2) an activation phase which appears as a simple diphasic potential and is designated P_{QRS}, and (3) a recovery phase, P_T, which is represented by a slow low-amplitude deflection.* P_{QRS} averaged about 3.2 millivolts in

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*The preauricular deflection P_p (or "O") which is occasionally present in examples of human endocardial electrocardiograms has been described for human heart by one of us and may also be seen in records published by Sodi-Pallares.[5] It is tempting to assign this deflection to the excitation of the sinus node. It is recalled that in cold-blooded animals the bulbus venosus gives rise to a striking preauricular wave and that a distinct deflection preceding P has been described for the mammalian electrocardiogram in leads taken from the vicinity of the sinus node.[18, 19]

height and 65 microseconds in duration. The deflection P_{QRS} representing auricular activation is similar to experimental records from the exposed auricular muscle of the dog obtained with a unipolar recording system by Wilson, Macleod, and Barker[10] and to the tracings of Craib from excised turtle heart muscle, striated muscle, and nerve.[9] The human record under discussion is contrasted in figure 2 with direct unipolar leads from frog hearts showing a spontaneously beating bulbus venosus (*C*, *D*) and auricle in situ (*B*), an electrically stimulated piece of auricular muscle (*E*), and (*F*) a strip of spontaneously beating ventricular muscle. Preparations *E* and *F* were submerged in turtle ringer solution. The similarity of the animal records to the human tracing is inescapable.

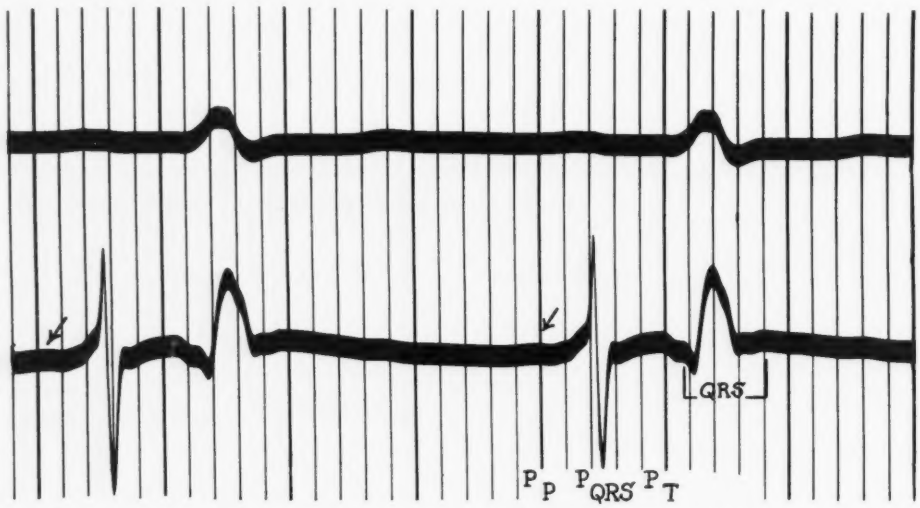

FIG. 1.— A human auricular electrogram. Upper tracing, Lead I; lower tracing, endocardial electrocardiogram from interauricular septum. Arrow points to small preauricular deflection (P_p). Ventricular QRS complexes characteristic of right bundle branch block. Time lines 0.040 second. (See text.)

THEORETIC CONSIDERATIONS

Because of its syncytial nature human auricular muscle may be regarded as if it were a large single cell. An exciting impulse arising at the sinus node exceeds the threshold of the surrounding auricular muscle and a wave of excitation spreads radially from the center of excitation. The wave of excitation invades the polarized, resting muscle and leaves behind a region of depolarized tissue. The tissue is restored to its normal resting stage by a wave of repolarization which does not necessarily and presumably in normal heart muscle seldom arises from the same focus of origin as the excitatory wave. This wave of excitation may be considered as a band in which the membrane is being depolarized according to the law of excitation. The band is not sharply demarcated but shades from complete polarization on the leading edge to complete depolarization on the following edge. Its effective width is appreciable and may range from a few millimeters to several centimeters. Since closely adjacent areas are in electrically different states (polarized or depolarized) a voltage difference of about 50 to 100 millivolts will exist between the two regions[20] and consequently a current will flow. For the sake of simplicity and without inducing a large error it can be assumed that the current flows between two lines one at the leading edge and one at the following edge of the band of depolarizing activity. This is equivalent electrically speaking to two line charges of opposite sign the effective width of the band apart and submerged in a volume conductor.

The two line charges may be considered to be formed of an infinite number of dipoles placed side by side (see fig. 5). It must be emphasized that the symbol is not the reality and that the dipole concept is only a convenient symbolization of the membrane theory of activation.

The two line charges when submerged in the volume conductor of the body fluids give rise

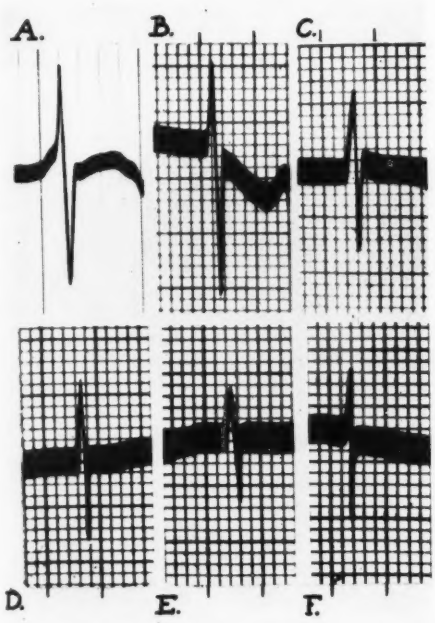

FIG. 2.—Direct leads from cardiac muscle. Exploring electrode in contact with cardiac muscle; indifferent electrode in *A* on central terminal, in *B*, *C*, and *D* on left leg, in *E* and *F* submerged in Ringer's solution distant from the source of current. *A*, Human electrogram of figure 1. *B*, Electrogram of auricle of frog in situ. *C* and *D*, Electrogram of bulbus of frog in situ. *E*, Record of strip of auricular musculature (frog) submerged in Ringer's solution and electrically stimulated. *F*, Unipolar record of strip of ventricular musculature submerged in Ringer's solution, beating spontaneously. Time lines 0.04 second.

to a current flow and to the accompanying lines of equipotential distributed according to the laws of volume conduction.

For auricular muscle a simple geometrical picture of the voltage lines about the source and the sink may be constructed. Such a representation, familiar since Waller's original discussion, is shown in figure 3. This figure demonstrates the lines of equipotential about two line charges in a conduction medium whose dimensions are large compared with the distance between the source and the sink. The two line charges and their equipotential lines extend throughout the conducting fluid of the body. Midway between the two charges is a line of potential which is equal to half of the voltage difference between the two. This line is often referred to as the zero line and is used as a reference for measuring the other voltages. If an electrode is placed in or on a volume conductor but at an appreciable distance from the source and the sink it will be effectively on, or very close to, the zero line (a "neutral" or "indifferent" electrode). An examination of figure 3 will show the truth of the statement. If now another electrode is placed so that the field about the line charges moves under or very close to this electrode, or if the electrode is moved along the line S-S from the extreme right of figure 3 to the extreme left, a diphasic wave will be recorded. In figure 4 a diphasic wave that was obtained in this fashion is illustrated. The resemblance to the diphasic action potential of figure 1 reproduced beside it is obvious and it is reasonable to believe that the P_{QRS} deflection of figure 1 arose from some such mechanism.

Figure 5 is a schematized version of two line charges viewed from above and passing across an exploring electrode with the other (neutral) electrode situated between the two charges. Again it is obvious that the passage of current over the electrode results in a plus-minus deflection. The distance between the two peaks of the recorded tracing of a direct lead represents the distance between the source and the sink and defines the width of the excitation wave, i.e., the band of tissue undergoing depolarization. The rapid downstroke of a record such as is illustrated in figure 1 may therefore be taken as an approximate measure of the width of this band. It is roughly identical with the so-called intrinsic deflection of a direct unipolar electrocardiogram. Given a rate of transmission of the action current through auricular muscle of 1,000 mm. per second,[22] the width of the band or the distance between the

source and the sink in the record presented here would approximate 10 millimeters. If recent figures on the rate of conduction through the human heart are substituted,[23] the width of the band would measure 4.5 to 5 millimeters. The width of the band in a dog's auricle calculated by Wilson, Macleod, and Barker measured 3.6 to 4 millimeters.[10] It must again be remembered that these values are only first approximations and that the actual width of the band of depolarization is indefinite.

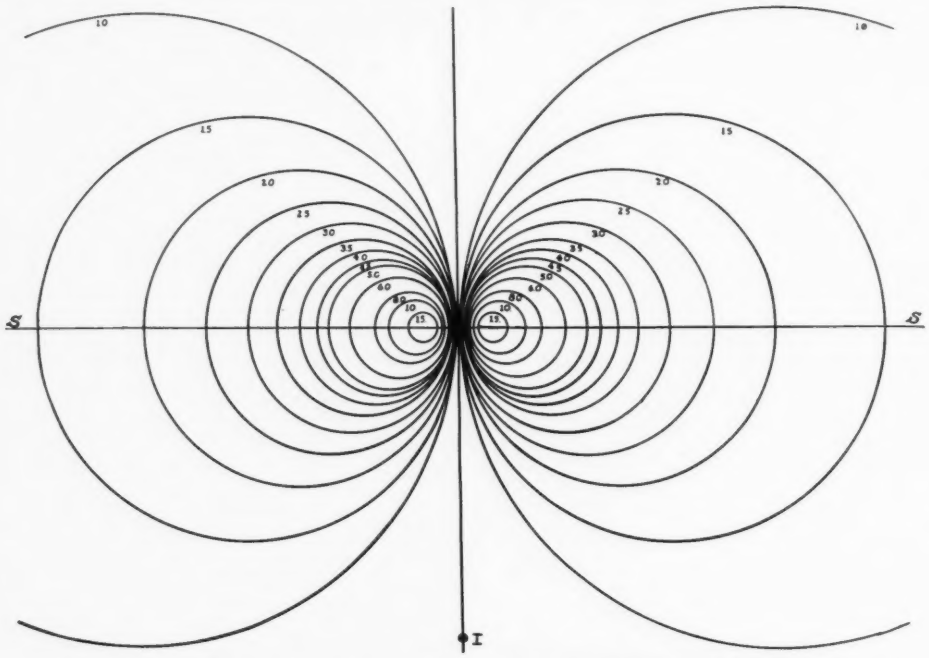

FIG. 3.—Concept of dipolar excitation. End-on view of equipotential lines about two small cylinders with a voltage difference between them and submerged in an infinite volume of conducting medium. S-S represents extension of a line connecting the center of the two cylinders; I, the voltage line halfway between the sink and the source which is used as a reference. If the diagram is considered to represent the voltage field about the sink and the source on an excitable cell, S-S would represent the cell surface, I the effective position of a distant ("indifferent") electrode. The figure is plotted from laws governing the flow of current in volume conductors (Knight and Fett[21]).

PRACTICAL CONSIDERATIONS

The sudden reversal of voltage as the source and sink pass the electrode has been termed the "intrinsic deflection" by Lewis, Meakins, and White.[22] If the exploring electrode is located at a place distant from the source and the sink but so oriented that the fields surrounding the source and sink cross the electrode, a similar diphasic wave will be obtained though of lesser amplitude. The peak-to-peak deflection of records of this type (semidirect leads) is known as the "intrinsicoid" or "semi-intrinsic" deflection[24] and, insofar as it can be interpreted as indicating the passage of the source and sink beneath the exploring electrode, serves as a valuable diagnostic aid.

The conclusion, however, that the intrinsicoid deflection is always the same as the intrinsic deflection must be viewed with caution. When the electrode is directly on the surface of excitable tissue, effects of adjacent areas undergoing excitation are small. With the electrode at some distance from the source and sink, other areas of excitation will materially

and appreciably contribute to the field at the electrode and the result represents the sum of all fields at this point. The observed intrinsicoid deflection may thus not represent the actual passage of the wave of excitation. This appears to be one of the reasons for the absence of a delay in the intrinsicoid deflection over the right ventricle in certain examples of right ventricular hypertrophy and for the apparent alteration of excitation over the right ventricle in instances of left ventricular hypertrophy and left bundle branch block. However, when the electrode is close to excited tissue, and when this tissue is belatedly activated, the intrinsicoid deflection is probably a valid indication of the passage of the excitatory wave.

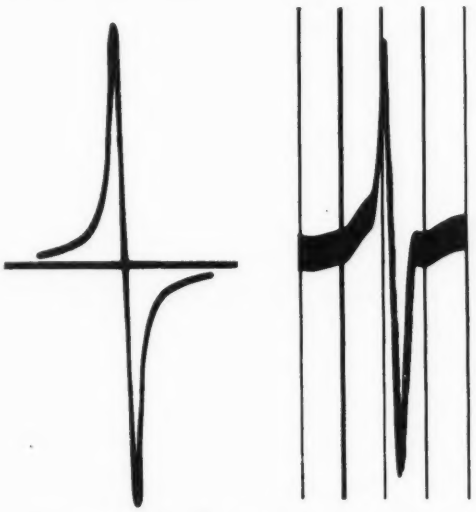

FIG. 4.—A comparison of calculated and recorded action potentials. *A* (left), artificial action potential calculated by placing an electrode on I of figure 3 and moving the other electrode along the line S-S from the extreme right of the diagram to the extreme left. The voltage lines crossed are plotted as a function of the distance along S-S and according to the laws of volume conduction. (See text.) *B*, Enlarged P_{QRS} of figure 1. The essential similarity to *A* is apparent.

Macleod, Wilson, and Barker[24] were unable to obtain an intrinsicoid deflection from auricular muscle comparable to that observed in leads from the vicinity of ventricular musculature. They suggested that this was due to the difference in the manner in which the excitation process spreads over ventricular as compared to auricular muscle. Deflections similar to the one under discussion, however, and clearly showing an intrinsicoid deflection have since been demonstrated with an electrode placed in the esophagus and almost in contact with left auricular tissue. A semi-intrinsic deflection is observed with such electrodes even when auricular flutter is present. Auricular endocardial records, not necessarily in contact with cardiac tissue, are likewise characterized by a diphasic component. The similarity between endocardial auricular and esophageal (epicardial) auricular records is in contrast with the striking difference that exists between records taken from the ventricular cavities when compared with epicardial ventricular leads. This confirms the theory that the order of excitation of auricular muscle, though not excitation itself, is different from that of the ventricles. In the auricles, excitation spreads radially from the sinoauricular node over the sheet of auricular tissue; in the ventricles, direct penetration of ventricular muscle by the excitation wave from within outward and its simultaneous emergence at the surface at several points are generally accepted.

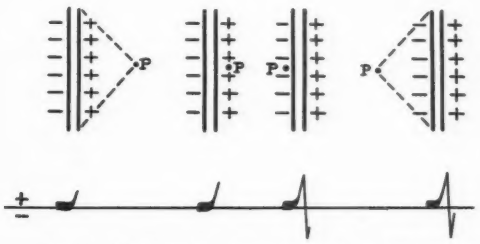

FIG. 5.—The dipolar concept of cardiac action currents and the intrinsic deflection. P defines a point representing the location of the exploring electrode. Electrical potentials recorded at P will show increasing positivity as the action current advances in the direction of P. As the dipole passes over P the potentials at this point will swing from maximum positivity to maximum negativity and the distance between the peaks will be proportional to the width of the dipolar band of cardiac accession. The interconnecting line between the two maxima of the curve is closely related though perhaps not identical with Lewis' intrinsic deflection.

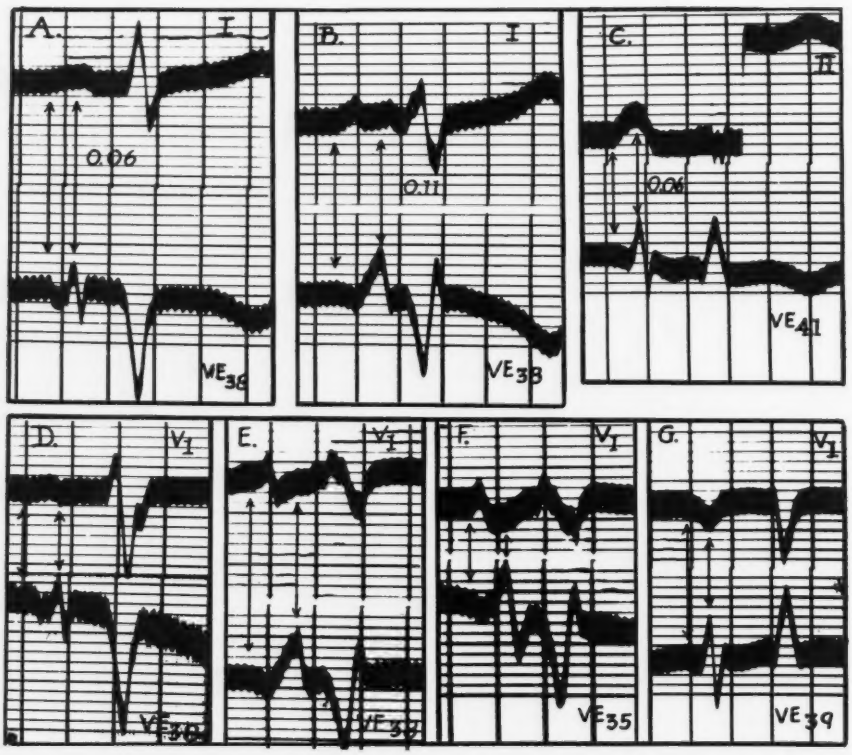

FIG. 6.—Semi-intrinsic deflection of esophageal leads. *A* and *D* represent esophageal leads of a normal subject obtained with an electrode placed 38 cm. from the front teeth and recorded simultaneously with Lead I (*A*) and Lead V_1 (*D*). Records *B*, *E*, and *F* are similar records of a 23 year old patient with advanced mitral stenosis and excessively striking "mitral P waves" (*B*). The esophageal electrode was placed 38 cm. (*B*, *E*) and 35 cm. (*F*) from the front teeth. Records *C* and *G* are those of a 59 year old man with advanced chronic obstructive emphysema. Unusually tall "pulmonary" P waves are displayed in the standard limb leads II and III (*C*).

The first arrow of each record defines the onset of auricular activation; the second the onset of the auricular intrinsicoid deflection. The figure (in seconds) denotes the onset of this deflection or the distance between the two arrows.

The onset of the intrinsicoid deflection in auricular esophageal leads of normal individuals is definitely later than the major portion of P in standard limb leads or in V_1 (*A*, and *D*). It is markedly delayed in *B*, *E*, and *F*, where definite left auricular enlargement was present, and occurs simultaneously with the second hump of P in I (*B*) and with or after the negative phase of P in V_1 (*E* and *F*). The esophageal record in the subject displaying P pulmonale appears normal in the face of an abnormal P wave in V_1. The onset of the intrinsicoid deflection and the shape of the preintrinsic plus deflection (P_R) in mitral stenosis is reminiscent of ventricular curves in bundle branch block. An early negative component (P_Q) is noted in all esophageal leads and coincides with the major and early upright component of P in Leads I and V_1.

The esophageal record displaying the latest intrinsicoid deflection of *F* is still definitely later than the onset of this deflection in *D* and *G*.

Film speed 50 cm. per second; time interval, 0.1 second.

Auricular esophageal leads reveal an intrinsicoid deflection which may be assigned to the activation of left auricular tissue. No intrinsicoid deflections other than those obtained from the endocardium by blood contact have ever been demonstrated for the right auricle. The true intrinsic deflection from this chamber apparently is hidden by the potentials from other

regions because of the distance of the electrode from the auricular surface. A lead usually considered to represent the effects of activation of the right auricle (V_1) shows a small, upright, rounded deflection quite different from the deflection of the left auricular esophageal lead (figs. 6 and 7). It may be argued that the absence of a distinct diphasic deflection obtained from an electrode in this position indicates that of excitation never completely passes under the region being explored.[2, 3] A chest lead in Lead V_1 position, usually directly opposite the right atrium, need not necessarily be considered a semidirect auricular lead. It is of interest, however, that the onset of right auricular activation, evidence of which appears simultaneously in leads from the right auricular endocardium and in V_1, definitely precedes the onset of

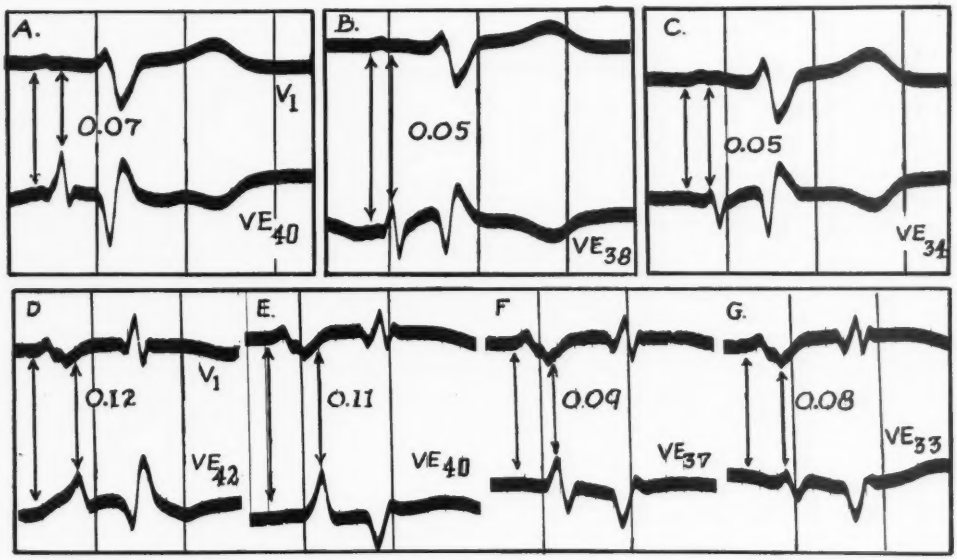

FIG. 7.—Normal and abnormal activation of left auricular musculature. Lead V_1 and esophageal auricular leads recorded simultaneously. *A*, *B*, and *C* were obtained from a 34 year old normal subject; *D*, *E*, *F*, and *G* from a 59 year old man with a markedly stenotic and calcified mitral valve, left auricular and right ventricular hypertrophy and dilatation (autopsy). The onset of the intrinsicoid deflection of all auricular esophageal leads in *D*, *E*, *F*, and *G* is definitely delayed and coincides with the final segment of the typically biphasic P wave in V_1. A single upright esophageal P deflection characterizes left auricular hypertrophy in the regions where the intrinsicoid deflection is most delayed while a biphasic P wave is usually seen in all auricular levels of the normal esophageal electrocardiogram. Film speed 75 mm. per second; time lines 0.2 second. Arrows and figures as in figure 6.

he electrode lies always in the field of one side of the dipole, that is, sharply to the left or to he right of the diagram of figure 3 and that he center line of the dipole never crosses the egion under the electrode. This would be possible if the impulse spreads radially over the uricular muscle. A rounded, upright P wave n V_1 would be so for the same reason that eads from the sinus region and from the area ephalad to it (upper auricular region and vena ava) are always uniformly negative: the wave positivity in left auricular (esophageal) leads of normal subjects (figs. 7 and 8) and that the intrinsicoid deflection recorded from these esophageal regions coincides approximately with the final portion of the small, rounded deflection of the P wave in V_1 or in any of the standard limb leads (figs. 6, *A*, and *D*, and 7, *A*, *B* and *C*).[25] It is exaggerated in left auricular hypertrophy and enlargement,[25-29] where a true delay in the intrinsicoid deflection similar to that observed over hypertrophied and di-

lated ventricular muscle, is common. In instances of this kind, V_1 or a lead taken one intercostal space higher, reveals a sharp, biphasic deflection remotely resembling the biphasic plus-minus deflection of figure 1 (figs. 6, 7, and 8). This was first noted by one of us[30] and has frequently been reported since. Standard limb leads usually reveal a broad and the electrode. Records of this kind should not be considered as representing an intrinsicoid deflection caused by hypertrophy of the right auricle which has frequently been proposed.[32-35] The biphasic pattern of the P wave in V_1 occasionally encountered in normal subjects may likewise be considered as a remote effect of auricular muscle distant from the exploring

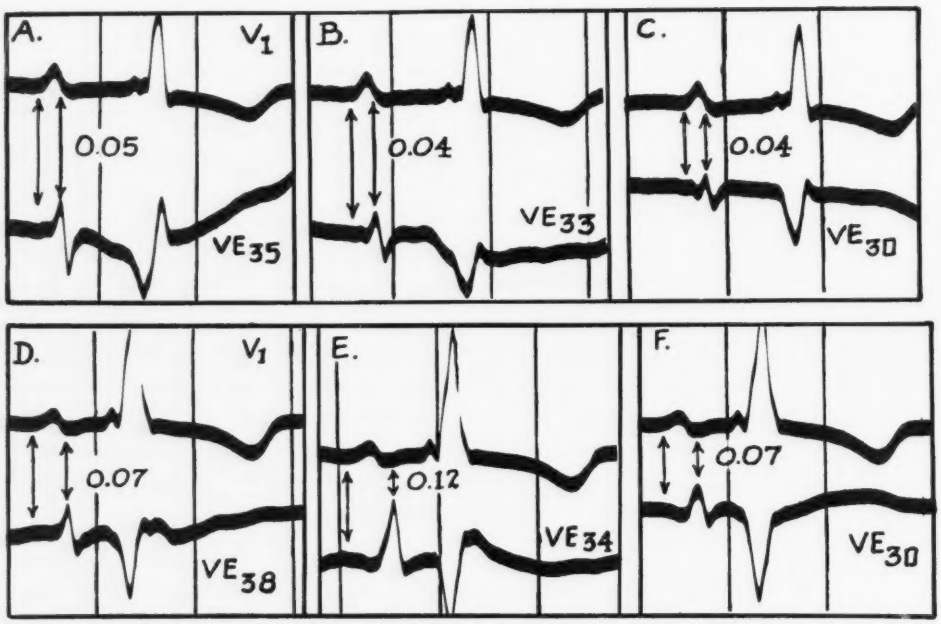

FIG. 8.—Right and left auricular enlargement with right ventricular hypertrophy. Lead V_1 and esophageal auricular leads recorded simultaneously. *A*, *B*, and *C* are from a 36 year old man with cor pulmonale following chronic pneumonitis and fibrosis of the entire left lung and of the lower and middle lobes of the right lung. The right ventricle was predominantly enlarged and the right auricle dilated (autopsy.) *D*, *E*, and *F* were obtained from a 24 year old man with advanced mitral stenosis. The electrocardiographic evidence of right ventricular enlargement was almost identical in both subjects. Auricular esophageal leads were normal in *A*, *B*, and *C* but showed definite delay of intrinsicoid deflection in esophageal leads with biphasic P waves in V_1 in the second case. Film speed 75 mm. per second; time lines 0.2 second. Arrows and figures as in figure 6.

bifid P wave in Lead I ("mitral" P of Wintern-itz[31]). The second hump of P in standard leads and the negative trough in V_1 coincide with the onset of the intrinsicoid deflection in left auricular leads (esophageal leads) (figs. 6, *B*, *E*, and *F*; 7, *D*, *E*, *F*, and *G*; 8, *D*, *E*, and *F*). It is assumed that precordial leads displaying a strikingly biphasic P wave in V_1 therefore express late activation of the left auricle and of auricular muscle remote from electrode. It is of interest to recall that the mechanical contraction of the right auricular muscle slightly precedes that of the left.[36]

Because no semidirect thoracic lead exists which reflects the changes of the right auricle, hypertrophy of this chamber appears almost impossible to diagnose electrocardiographically. Originally, a tall and upright P wave in Leads II and III was taken as evidence of right auricular hypertrophy ("P pulmonale"[31]). In clin-

ical examples of this kind, the P wave in V_1 is rarely altered (fig. 8, *A*, *B*, and *C*), though it may at times be negative or small and biphasic (fig. 6, *G*). Esophageal leads from such subjects fail to show a late activation of left auricular tissue (figs. 6, *C* and *G*, and 8, *A*, *B*, and *C*). It is therefore postulated that the pattern of P pulmonale is not caused by unilateral auricular hypertrophy but may be primarily the result of abnormal cardiac rotation or of auricular dilatation which allows the electrical effects of both auricles to be deflected backward and toward the left leg. The lack of

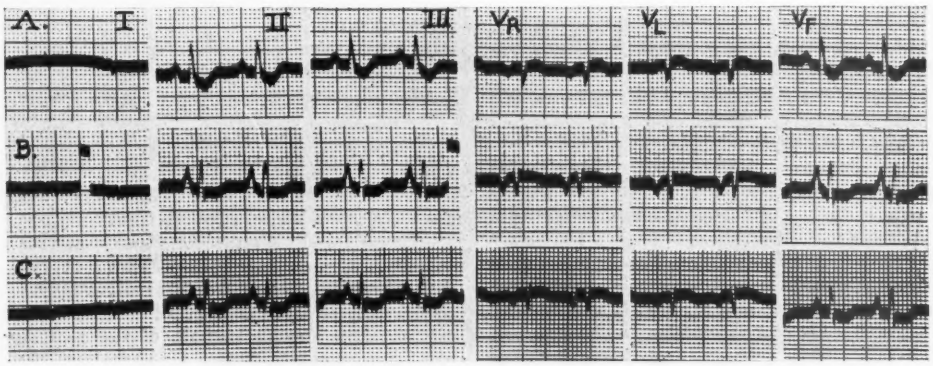

FIG. 9.—Transient pulmonic P waves. Records of R. B., a 56 year old man with far-advanced chronic obstructive emphysema. *A* was obtained on July 7, 1948, *B* on August 3, 1948, and *C* on August 11, 1948. Treatment with digitalis was discontinued on July 12, 1948, and the patient lapsed into congestive heart failure. Rapid redigitalization was instituted on August 4, 1948, which resulted in prompt compensation. "Pulmonic" P waves appear in Leads II and III during the period of failure only. This is mainly occasioned by a sharp increase in P in V_F. Distension of the right auricle rather than auricular hypertrophy is suggested as the explanation for the transient changes.

correlation between right auricular hypertrophy and "P pulmonale" has been frequently noted.[36-40] Figure 9 reveals an example of temporary acute cor pulmonale in a patient with chronic disease of the lungs. During the phase of failure, huge "pulmonary P waves" were present which promptly disappeared upon recovery. Observations such as these likewise argue against the concept that views P pulmonale as a manifestation of right auricular hypertrophy. On the other hand, regression of mitral P waves, once present, has never been observed to our knowledge. The two types of P-wave changes are therefore unrelated and arise from entirely different mechanisms.

CONCLUSIONS

1. Direct electrocardiograms obtained from the human auricular muscle in situ allow a reexamination of the dipole approximation of the membrane theory of cardiac excitation.

2. It is assumed that during activation a current flows between two lines, one at the leading edge and one at the following edge, of a band expressing depolarizing activity of muscle units submerged in a volume conductor.

3. The line charges and their equipotential lines extend throughout the conducting fluid of the body and an electrode located in the path of the moving dipole will record a diphasic plus-minus deflection. Employing available formulas for the flow of currents in volume conductors, a record may be plotted that has striking similarities to those obtained from direct leads in animals and man.

4. These considerations are directly applicable to semidirect auricular leads. An electrode placed in the esophagus may be considered to lie in the field of the moving dipole.

5. The intrinsicoid deflection of such leads may be used to estimate left auricular hypertrophy. From these the remote effects of auricular activation on standard limb leads and on

certain precordial leads can be interpreted with reasonable accuracy.

6. The "mitral" P wave and sharply biphasic P waves in V_1 may be considered to result from left auricular enlargement. Right auricular hypertrophy on the other hand cannot be diagnosed from the electrocardiogram and examples of "pulmonary" P waves may be caused by abnormal cardiac rotation allowing the effects of both auricles to be deflected backward and toward the left leg.

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Studies in Intracardiac Electrography in Man

IV. The Potential Variations in the Coronary Venous System

By Harold D. Levine, M.D. and Walter T. Goodale, M.D.
with the technical assistance of Eunice Gilman

Two parallel researches in progress at the Brigham Hospital, one an investigation of intracardiac electrography in general, the other a long-term study of heart muscle metabolism with the aid of the venous catheter, converge, resulting in the interesting practical and theoretical findings reported here. The authors show how the electrogram may help establish the position of the catheter in the coronary venous system and indicate the type of potential variations recorded in the coronary sinus, in its continuation, the great cardiac vein, and in its tributary, the middle cardiac vein.

UNTIL recently our concepts of the mode of electrical activation of the human heart have been derived largely from studies of the canine heart. With the advent of venous catheterization technic, and the possibility of recording the potentials within the right side of the heart, the similarity of right sided potentials in man to those in the dog has been demonstrated.[1–10] *Left* ventricular cavity potentials have also been recorded in man, once by the hazardous procedure of passing a catheter through the arterial system and the aortic valve into a normally activated left ventricle,[11] and once by passing the catheter through an interatrial septal defect and the mitral valve into the left ventricle of a patient with right bundle branch block.[12] In both of these studies, the QRS complex within the left ventricle was directed downward. Since in both instances the septum is presumed to be activated in a normal direction from its left to its right side, these studies apparently confirm the similarity of left as well as right ventricular cavity potentials in man to those in the dog. In two other studies,[7, 9] the recording of left-sided potentials was not clearly documented.

Coronary venous catheterization technic[13] has offered still another feasible approach to the study of the electrical activation of the human heart, as we have already reported in preliminary form.[14] This procedure has been used chiefly for studies on coronary blood flow and myocardial metabolism in dogs and man, in collaboration with Bing, Eckenhoff and their associates.[15–18] One observation of the electrical potentials recorded through a catheter fortuitously inserted into the coronary sinus of a patient with hypertensive cardiovascular disease was reported recently by Kert and Hoobler.[10] The present report is an analysis of the potentials recorded through a catheter systematically inserted into the various accessible branches of the coronary venous system, including the coronary sinus, great cardiac vein and middle cardiac vein, among six subjects with essentially normal hearts

Method

The coronary sinus, great cardiac vein or middle cardiac vein were catheterized using a No. 7 or 8F "bird's eye" venous catheter provided with multiple openings near the tip, as described elsewhere.[18*]

A systematic technic of intubating the coronary sinus and its branches, following the essential principles formulated for the same procedure in intact

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* Available from the U. S. Catheter and Instrument Co., 334 Bay St., Glens Falls, N. Y.

dogs,[15] was applied to the present studies in man. The regional anatomy pertinent to coronary sinus catheterization was recently described by Bing and associates,[16] and will therefore not be reviewed here.

The catheter was introduced into the right atrium by the technic of Cournand and Ranges.[20] The tip was then passed into the inferior vena cava. This maneuver was often aided by having the patient take a deep breath just as the catheter was advanced downward from a position low and posterior in the right atrium. Often the right anterior oblique position gave a better view of the posteriorly oriented inferior vena cava, so that the catheter could be directed into it more deliberately.

Once inside the inferior vena cava, with the subject now returned to the posteroanterior position, the slightly curved tip was turned to point medially (toward the patient's left), and cautiously withdrawn into the atrium. At the instant at which the catheter tip flicked free from the vena cava into the atrium proper, a slight medial shift of the tip occurred, aided by the fact that the curved tip had already been directed medially. This slight shift carried the tip to a point directly opposite the coronary sinus ostium, which lies immediately adjacent to the inferior vena cava, on the posteroinferior medial surface of the right atrium, with only the Eustachian ridge separating the ostia of the two vessels. After several thrusts of the catheter at this point, the tip would usually enter the coronary sinus ostium. Again, alternating posteroanterior and right anterior oblique fluoroscopic views often helped to make sure that the catheter remained properly oriented along the posterior atrial surface. If the tip was allowed to drift anteriorly, it would almost invariably pass through the tricuspid valve into the right ventricle.

As the catheter now passed into the coronary sinus and on into the great cardiac vein, it appeared to turn sharply across the cardiac shadow, slightly cephalad, across the posterior surface along the atrioventricular groove, often reaching the left border of the heart. Occasionally the catheter entered the middle cardiac vein, passing from its ostium, just inside the coronary sinus ostium, out along the posteroinferior septal surface toward the apex.

Difficulty in intubating the coronary sinus because of deflection of the catheter by a prominent Eustachian ridge has been reported.[16] This has not been apparent during the present study, possibly because of selection of a catheter with only a slightly curved tip. The optimal curve in the tip for this procedure has appeared to be much less than the optimal curve for entering the right ventricle and pulmonary artery. Likewise the remnant of the valve of the coronary sinus (thebesian valve) has never appeared to interfere with coronary sinus intubation in the adults studied. Insertion into the coronary sinus has been found to be distinctly easier from the left arm, because of the natural tendency for the curve to be oriented medially in the right atrium when approached from this direction. It has sometimes been difficult or impossible to keep the tip pointed in the proper direction on withdrawal from the inferior vena cava, when the heart has been approached from the right arm. It has also been very difficult to approach the coronary sinus with any degree of consistency by first going through the tricuspid valve and using this as a point of reference rather than the inferior vena cava. It has usually been obvious within two or three minutes of fluoroscopy time whether or not the catheter has been properly curved to enter the coronary sinus. If not, a second, third or even a fourth catheter has been tried in succession, often with success as one with the proper curve and orientation has been obtained. Seventeen out of 30 such procedures have been technically successful over the past year, with most of the failures occurring in heavy or emphysematous patients where fluoroscopic visualization has been poor. Patients with dilated or hypertrophied left ventricles have presented no special difficulty by the technic described, possibly because such patients also tend to have a dilated coronary sinus. Once the catheter has been passed successfully into the inferior vena cava, the technic of coronary sinus catheterization has been successful in 7 of the last 11 procedures.

The localization of the catheter within the coronary venous system was determined first by the characteristic course of the catheter under fluoroscopic control. Distinction between a position within the right ventricle and the coronary sinus was easier in the right anterior oblique than in the posteroanterior view, as illustrated elsewhere in dogs.[15] Successful coronary venous catherization was then confirmed by the pressures recorded through the catheter by means of a Sanborn electromanometer. Unless there was some element of coronary venous obstruction by the catheter, there was never more than 1 or 2 mm. pressure gradient between the mean levels in the coronary venous system and the right atrium (see table 1), and a characteristic short systolic pulse pressure wave was clearly distinguishable from pressures in the right ventricle or right atrium. Absence of extrasystoles did not prove to be a completely reliable indication that the catheter was in the coronary venous system rather than the right ventricle, as discussed below, but it was none the less true that rapid runs of multiple ventricular extrasystoles were seen only when the catheter was in the right ventricle. Finally, coronary venous blood samples were much darker than mixed venous blood, as emphasized in previous reports, reflecting a very low coronary venous oxygen saturation. Significantly lower levels of pyruvate, lactate and glucose were also characteristic of coronary venous blood compared to arterial blood, measured

by methods discussed in detail in connection with similar findings in dogs.[15] (See table 1.)

A stainless steel wire of 1/64-inch diameter was passed to within four centimeters of the tip of the catheter. The proximal few centimeters of the wire were incorporated in and passed through a three-way stopcock whose distal end fitted into the proximal end of the catheter and whose lateral arm received the tube leading to a saline drip apparatus or a Sanborn electromanometer (condenser microphone type). The proximal end of the wire was coupled to a central terminal of the Wilson type. A continuous saline drip was run through the side arm of the adapter except when pressure measurements were being made or samples of blood were being drawn for chemical analysis. To prevent the development of artefacts and wandering of the baseline of the electrogram, it was necessary to eliminate any leakage about the adapter. Tracings of the intracardiac potentials were recorded simultaneously with the unipolar left leg lead (V_F) with a Sanborn Tri-Beam Electrocardiograph. In one instance a Cournand double-lumen catheter,[21] whose proximal lateral opening was 10 cm. from its distal terminal opening, was used. In this way it was possible to record the potentials simultaneously at two points within the heart, e.g., coronary sinus and right atrium, pulmonary artery and right ventricle, or right ventricle and right atrium. In all cases additional recordings were made at one or more points in the right ventricle and atrium, as well as conventional and unipolar limb leads and unipolar chest leads for comparison with the potentials in the coronary venous system.

TABLE 1.—*The Chemical and Manometric Findings in Coronary Venous Blood*

Name	G. L.	B. C.	G. C.	T. D.	A. B.
Pressures, mm. Hg					
Coronary venous:	7	(8*) 5	2	(12*) 5	(15)*
Right Auricle:	6	5	3	4	1
Right Ventricle:	24/7	28/5	30/0	—	—
Oxygen Contents, vol. %					
Arterial:	16.7	15.2	17.2	12.1	16.9
Coronary venous:	5.6	4.7	6.7	3.7	5.2
Pyruvate, mg. %					
Arterial:	1.2	1.4	1.2	2.1	—
Coronary venous:	0.8	0.7	1.0	0.9	—
Lactate, mg. %					
Arterial:	6.8	5.1	10.5	12.4	—
Coronary venous:	4.1	3.0	7.1	5.9	—
Glucose, mg. %					
Arterial:	110	92	106	80	—
Coronary venous:	98	81	97	74	—

* Values in parenthesis were apparently obtained with the catheter partially obstructing the coronary venous system, with lower subsequent values obtained after withdrawing the catheter slightly.

RESULTS

Seven successful coronary venous catheterizations were carried out in 6 individuals with normal hearts, among 12 deliberate attempts in 10 subjects. In 5 of these subjects the potentials of the coronary sinus and great cardiac vein were recorded. In 2 subjects records were obtained with the catheter at several different points within the middle cardiac vein.

The Potentials in the Coronary Sinus and Great Cardiac Vein

In subject G.L., when the catheter was in the great cardiac vein the auricular complex was directed entirely upward (fig. 1*A*) with the intrinsic deflection at 0.08 second. The descending limb did not quite return to the isoelectric line but was followed by a secondary upward deflection (R′a) with a slightly more gradual slope, measuring about half as tall as Ra. The returning limb of the secondary R wave descended slowly and flattened out to form an upward deviation of the auriculoventricular segment (R′a-Q) in the manner described elsewhere.[3, 8] This gave way to a deep broad (0.06 second) ventricular Q wave, a less prominent R wave, and an inverted T wave. When the catheter was now withdrawn about 2 cm. into the coronary sinus proper, the auricular deflection (fig. 1*B*) consisted initially of a slow upward component beginning about 0.04 sec-

nd after the beginning of the P wave recorded imultaneously in Lead V_F. This was superseded by a rapid equi- and bi-phasic deflection (RSa) whose apex (intrinsic deflection time, recorded at the apex of this rapid auricular deflection) occurred 0.07 second after the beginning of the P wave. The Sa wave returned not quite to the isoelectric line and was superseded by a small downward deflection which followed immediately after the completion of the P wave in Lead V_F. This in turn was followed by a biphasic ventricular complex consisting of a prominent Q wave 0.06 second in duration and exceeding the following ventricular R wave in both magnitude and duration. This was followed by an inverted T wave.

In this individual, electrograms were also obtained at high and low levels in the right atrium. It will be noted that at low levels (fig. 1*C*) the intrinsic deflection occurred at 0.03 second and at high levels (fig. 1*D*) at 0.02 second. The ventricular complex at each level consisted of a broad QS followed by an inverted T wave. It was also observed that whereas deep inspiration moved the catheter a considerable distance and produced corresponding changes in the appearance of the auricular complex while the catheter was in the right atrium, a similar deep inspiration while the catheter was in the great cardiac vein produced no change in electrographic appearance or in the position of the catheter as determined by fluoroscopic observation in posteroanterior and oblique projection. This is interpreted as evidence of the absolute or relative immobility of the catheter while in the great cardiac vein, the force developed by inspiration apparently merely taking up the slack in the intra-atrial portion of the catheter.

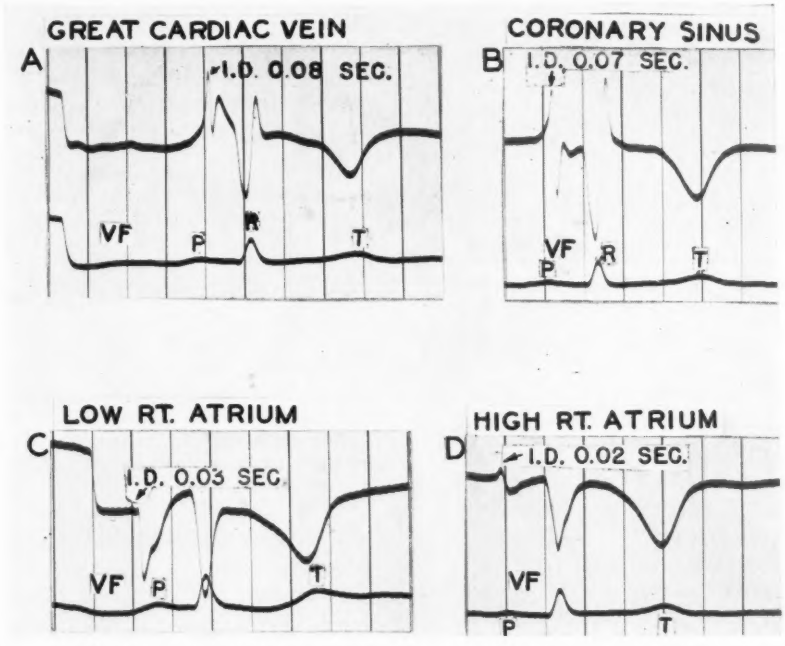

FIG. 1.—Subject G. L.: Potentials in great cardiac vein (*A*) and coronary sinus (*B*) compared with potentials low (*C*) and high (*D*) in the right atrium. Note the lateness of the intrinsic auricular deflection in the coronary venous system as compared with the right atrium, and the prominent ventricular Q and R in the coronary veins compared with the QS type of deflection in the right atrium.

In subject G.C. electrograms were recorded at only one point in the coronary sinus. At this location the auricular complex (fig. 2) con-

sisted of an upward deflection (Ra) with an intrinsic deflection at 0.07 second. The returning limb of this Ra was interrupted by a sudden break with a slower return toward the isoelectric line. This constitutes an upward deviation of the Ra-Q segment. This in turn was superseded at a level still above the isoelectric line by a prominent Q wave which began shortly before the beginning of Q of simultaneously recorded Lead V_F. The broad Q was followed

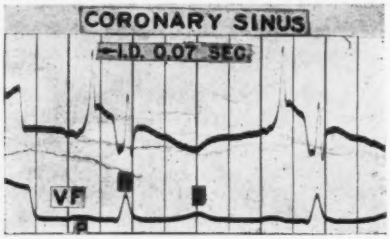

FIG. 2.—Subject G. C.: Characteristic coronary sinus potentials (upper curve) with a very tall upright P wave, late intrinsic auricular deflection (0.07 sec.), prominent Q and R waves. Simultaneous Lead V_F (lower curve).

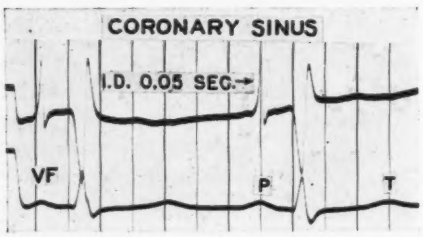

FIG. 3.—Subject C. M.: Coronary sinus potentials (upper) with simultaneous V_F lead (lower curve). Intrinsic auricular deflection at 0.05 sec.; deep Q wave, prominent R wave.

by a prominent R, not quite so large as Q, and this in turn by a prominent S wave. The T wave was inverted and asymmetrical with a long gradual descending limb and a more rapid ascending limb. Tracings taken high in the right atrium showed the intrinsic deflection to occur at 0.01 second.

In subject C. M., electrograms again were obtained at only one point in the coronary sinus. In this instance the auricular complex (fig. 3) began with a small rounded elevation synchronous with the first half of P_{VF} interrupted by a large Ra wave, which in turn was followed by a small Sa wave. The intrinsic deflection of the auricular complex occurred at 0.05 second. After an isoelectric interval, this was followed by a broad Q wave (0.05 second in duration) and this, in turn, by a slightly smaller and narrower R wave. The T wave was notched and slightly inverted.

In subject T.D., the coronary sinus tracings were somewhat atypical (fig. 4). The Ra deflection was much less conspicuous. The intrinsic deflection was inscribed at 0.07 second. The Q wave was less prominent and narrower (0.03 second) and R more prominent than in the coronary sinus tracings described above.

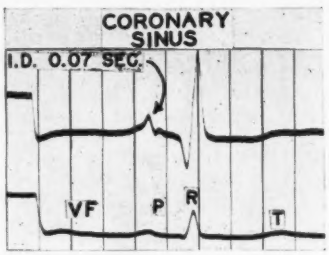

FIG. 4.—Subject T. D.: Coronary sinus potential with small intrinsic deflection at 0.07 sec., prominent Q and tall R wave.

The T wave was biphasic but predominantly inverted.

In subject M.C., tracings obtained in the great cardiac vein (fig. 5*A*) showed a very large auricular deflection with the apex of Ra at 0.06 second. A definite Sa wave followed. This was followed by large ventricular Q and R waves and a biphasic T wave.

On withdrawing the catheter from the great cardiac vein to the coronary sinus proper numerous supraventricular premature beats developed and the Sa wave disappeared (fig. 5*B*). The intrinsic deflection was now inscribed at 0.07 second. Meanwhile, although ventricular Q and R waves were retained, an auricular S wave reappeared (fig. 5*C*, beginning of strip). Continuous tracings (fig. 5*C*) were then recorded as the catheter was withdrawn from the coronary sinus into the right atrium. Reference to this figure shows how the complexes were characteristic successively of the coronary

sinus, then of the right atrium and finally of the right ventricle.

sec.) electrograms from the first 4 subjects described above are reproduced in figure 6. The

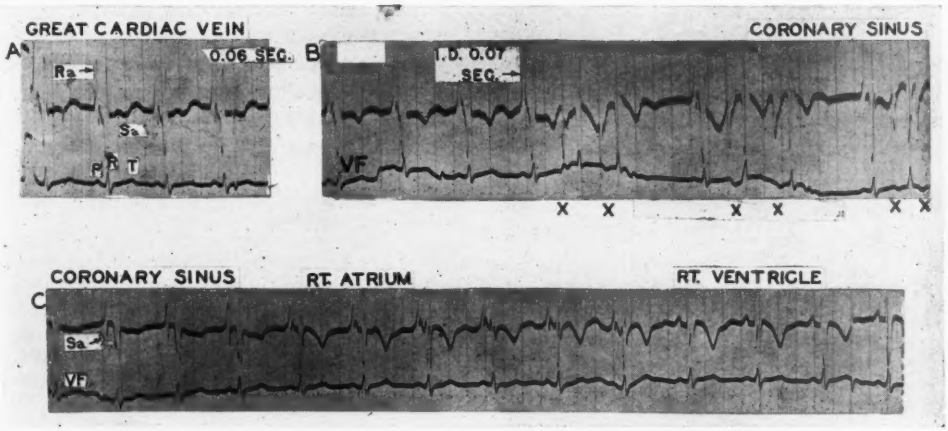

FIG. 5.—Subject M. C.: Excerpts from a continuous tracing during withdrawal of the catheter from the great cardiac vein. An Sa wave is present deep in the great cardiac vein (*A*), disappearing at a point (*B*) in the coronary sinus, only to reappear in the proximal coronary sinus (*C*). The final strip (*C*) was recorded as the tip of the catheter was withdrawn from the coronary sinus into the right atrium and passed into the right ventricle, and shows in turn the characteristic potentials of each region. Note the supraventricular premature beats (x) in strip *B*, during withdrawal of the catheter through the coronary venous system.

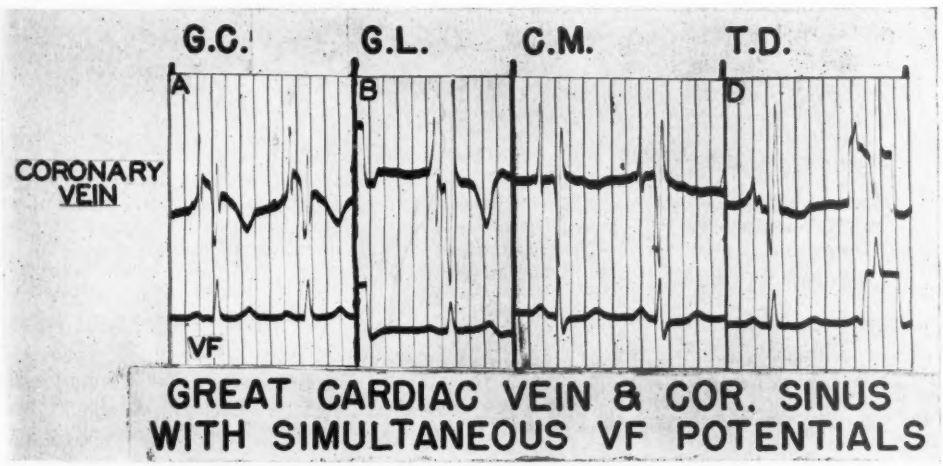

FIG. 6.—Low speed electrograms (25 mm. per sec.) from the coronary venous system (upper) with simultaneous V_F potentials (lower curves) in 4 normal subjects. Note the family resemblance of these tracings.

To demonstrate the family resemblance of potentials recorded in the coronary sinus or great cardiac vein, the low speed (25 mm. per

existence of auricular intrinsic deflections, usually of considerable magnitude, their inscription at the height of or during the latter half

of the P wave of simultaneously recorded Lead V_F and the existence of prominent ventricular Q and R waves and of inverted ventricular T waves are at once apparent.

The Potentials in the Middle Cardiac Vein

In 2 of the 6 individuals studied, the catheter was passed into the middle cardiac vein as determined by fluoroscopic examination and roentgenograms made with the subject rotated in various positions as well as by chemical and manometric determinations (table 1). In patient A.B., with the catheter a considerable distance into the vein, a flat P wave (fig. 7*A*), followed by an R wave, was recorded. No Q wave was present. The T wave was upright. These tracings correspond to left ventricular epicardial potentials. When the catheter was drawn a few centimeters more proximal, the electrogram (fig. 7*B*) changed abruptly from a predominantly upright to a downward (QS) complex followed by a flat T wave. A ventricular R wave was not recorded. This tracing probably represented right ventricular epicardial potentials.

The absence of a Q wave when the catheter was over the left ventricular epicardium and the absence of an R wave over the right ventricular epicardium suggests that the catheter tip may have been very close to the septum in each instance.

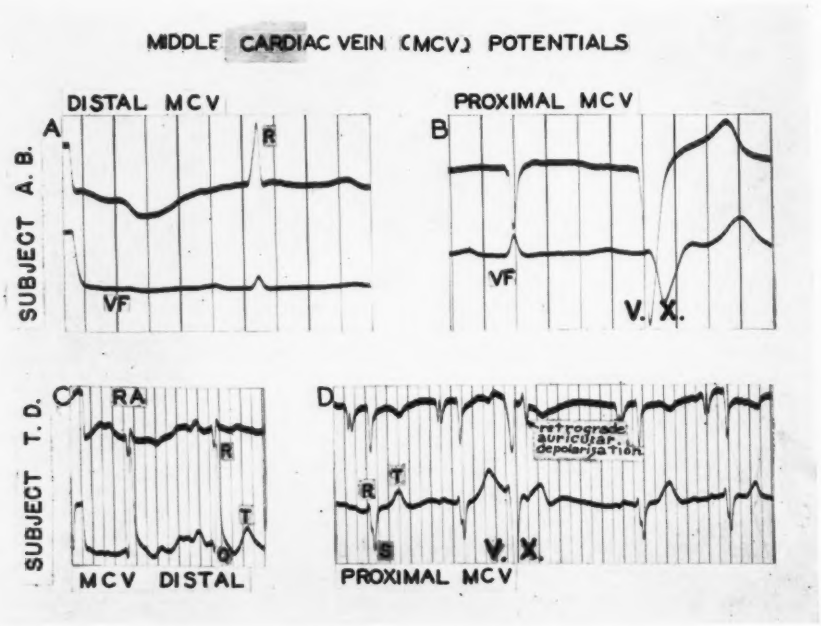

FIG. 7.—Electrograms recorded in the distal portion (*A* and *C*) and the proximal portion (*B* and *D*) of the middle cardiac vein (MCV) in 2 subjects. Note the development of ventricular premature beats with the catheter in place in each subject. In the lower tracings (*C* and *D*) a double lumen catheter was used, with the proximal lumen in the right atrium (RA).

Subject T.D. showed a similar left ventricular epicardial potential when the catheter was passed for 4 to 5 cm. into the middle cardiac vein, although here there was a small Q wave (fig. 7*C*, lower curve). On withdrawing the catheter more proximal again an abrupt shift to a right ventricular epicardial pattern was noted, this time with a small R wave (fig. 7*D*, lower curve). Since this R wave of the right ventricular epicardial potential coincided with the Q wave of the left ventricular epicardial potential, it seems reasonable that they represent two aspects of the same electrical event,

namely, septal activation. Furthermore, unlike the tracings in subject A.B., each of the electrode positions must have been far enough from the plane of the interventricular septum to face respectively the approaching wave of septal activation at the proximal position and the retreating wave at the distal position.

Isolated ventricular premature beats were noted when the catheter was passed into the middle cardiac vein in each instance as illustrated in figures 7*B* and 7*D*.

When figure 7*D* was recorded, the proximal opening of a double lumen catheter was in the right atrium near the superior vena cava (upper curve) and the distal opening in the middle cardiac vein (lower curve). The third QRS complex is premature and of greater duration and magnitude than the preceding or following QRS complexes. As recorded at the superior caval electrode, the auricular complex follows the premature ventricular complex, interrupting the ventricular T wave. This auricular complex, in contrast to all other auricular complexes in this strip, which were directed entirely downward, was biphasic with an initial upward and final downward deflection. This indicates that whereas the impulse in the normal beats proceeded away from the superior vena cava, in the premature beat atrial depolarization first approached then receded from the superior vena cava. The post-extrasystolic pause was quite compensatory but the changed direction of atrial depolarization suggests retrograde depolarization of the atrium if not of the sinoatrial node.

Discussion

Left Atrial Potentials

Previous studies[1–8] have demonstrated the presence of large auricular deflections within the right atrium, being inscribed early at high atrial levels and later at low atrial levels, but in general occurring during the first half of the P wave of a simultaneously recorded limb lead. By the use of simultaneously recorded intra-atrial and esophageal leads,[8] it was shown that the right atrium was activated some 0.05 to 0.08 second earlier than the left atrium as recorded in the esophageal lead at approximately the same horizontal plane. It appeared then that, in general, the right atrium is stimulated before the left and that the auricular intrinsic deflection occurs during the first half of the auricular complex in the right atrium and during the second half of the auricular complex in the left atrium.

When the catheter is introduced into the orifice of the coronary sinus in the lower part of the right atrium, it must take a leftward course. Unless the tip happens to enter the tributary middle cardiac vein, the catheter, on further insertion, tends to follow the course of the coronary sinus and its continuation, the great cardiac vein. This circles around the left side of the atrioventricular groove, then the left anterior aspect of this groove to the anterior longitudinal sulcus where it turns toward the cardiac apex, paralleling in a general way the course of the descending branch of the left coronary artery. After passing through the heart wall at the inferior angle of the right atrium, the catheter very promptly enters the atrioventricular groove. At this point the electrode is at the brim of the left atrium and of the left ventricle, and capable of recording the potentials of both of these cavities. This is illustrated in figure 8, which shows the heart in sagittal section. It will be obvious that an electrode in the coronary sinus would reflect the potentials not only of the left atrial and ventricular cavities, but also of a portion of the epicardial surface of the left ventricle.

One would predict then that the electrode in the coronary sinus must record the potentials of the lower part of the left atrium. The auricular intrinsic deflection should therefore occur at the height of or during the latter half of the P wave of simultaneously recorded Lead V_F. The further the electrode lies from the atrial pacemaker, the later should this intrinsic deflection be inscribed. Assuming uniform conduction of the impulse over the atrial surfaces, the electrode must eventually reach a point on the anterolateral aspect of the atrioventricular groove 180 degrees from the projection of the sinoatrial node upon this groove, toward which point the wave of atrial depolarization must converge from all angles. At this point the rapid auricular deflection should be entirely upright, for no atrial muscle is stimulated after this point. Figures 1*A*, 2 and 5*B* are actual examples of the type of electrogram predicted. At points nearer the cardiac pacemaker the rapid deflection should be largely upward (Ra) but this might be followed by a downward (Sa) deflection because some atrial

muscle, further from the pacemaker, is stimulated later. Actual examples of this theoretic potential are illustrated in figures 1*B*, 3, and 5*C*. If the catheter is now passed to a point beyond the meridian intercepting the pacemaker, the intrinsic deflection may again appear earlier and an Sa wave may reappear because atrial muscle is now activated later than this new point. This is borne out by figures 5*A–C* showing an Sa wave in the great broad Q wave followed by an R wave. This Q wave corresponds to the initial ventricular deflection recorded directly in the chamber of the dog's left ventricle.[4] It corresponds also to the initial part of the human left ventricular cavity electrogram described by Zimmerman and co-workers[11] in aortic insufficiency, and by Sodi-Pallares.[12] The evidence of the present study of normally activated hearts establishes this broad Q wave recorded in the coronary

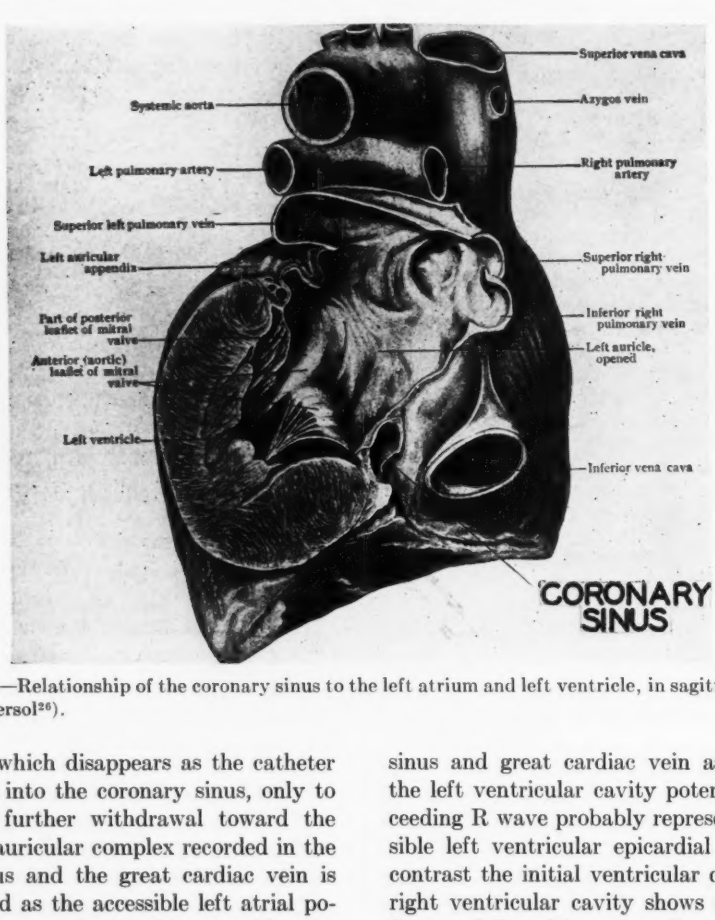

FIG. 8.—Relationship of the coronary sinus to the left atrium and left ventricle, in sagittal section (after Piersol[26]).

cardiac vein which disappears as the catheter is withdrawn into the coronary sinus, only to reappear on further withdrawal toward the atrium. The auricular complex recorded in the coronary sinus and the great cardiac vein is thus explained as the accessible left atrial potentials. In a general way this resembles the potentials recorded by the esophageal electrode at atrial levels.[22–24]

Left Ventricular Potentials

The initial ventricular complex in the coronary sinus and great cardiac vein begins as a sinus and great cardiac vein as representing the left ventricular cavity potential. The succeeding R wave probably represents the accessible left ventricular epicardial potential. By contrast the initial ventricular complex in the right ventricular cavity shows a small initial R wave followed by a deep S wave. The initial ventricular complex recorded in the right atrium generally consists of a QS deflection.

Ventricular repolarization is normally represented as an upright T wave on the precordium overlying the left ventricle. In our experience[9] and in that of most observers[3, 4] it

is usually recorded as an inverted T wave in the right ventricular cavity but occasionally has been reported as upright in that location.[7] Since the coronary sinus is normally in anatomical relationship with the left ventricular cavity and only to a slight extent with the left ventricular epicardium, the potentials recorded there must represent the composite effect of left ventricular cavity and epicardial potentials. Since the T wave is inverted in the coronary sinus, the cavity effect must be predominant. This relationship primarily to endocardium is emphasized in figure 8.

Generalizations are hardly warranted on the basis of the observations of the potentials in the middle cardiac veins of only two individuals. They do suggest some variability in the curves registered in that vessel depending possibly upon a variation in the course of this vessel in relation to the posterior interventricular sulcus. As the catheter in this vessel crosses the interventricular septum toward the apex, an abrupt change from predominantly downward (right ventricular) to predominantly upward (left ventricular) potentials may be detected as illustrated in figure 7.

Kert and Hoobler[10] published electrograms obtained from three positions in the coronary sinus of a patient with essential hypertension. Each electrogram showed auricular intrinsic deflections occurring during the latter half of the P wave recorded simultaneously in Lead V_F. The first of the three tracings showed a broad ventricular Q wave followed by a broad R wave; the other two showed very inconspicuous ventricular Q waves but very prominent R waves. These R waves differed in time from one position to another in the coronary sinus. The authors suggest that they reflect the activation of different portions of the base of the left ventricle. The discrepancy between these observations and ours, in which a broad Q wave was always recorded with the electrode in the coronary sinus, may be related to the fact that all of our subjects had normal hearts, whereas the patient of Kert and Hoobler had left ventricular enlargement. It is conceivable that under the latter circumstance the coronary sinus may lie in a more intimate relationship with the epicardial than the endocardial aspect of the left ventricle.

Accidental Catheterization of the Coronary Sinus

In a previous communication from this laboratory,[9] an initial ventricular R wave was noted in what was at first sight considered to be the right ventricle in all but 3 of 27 individuals. In 2 of these exceptions (subjects 6 and 26, figures 7*F* and 8 of that paper) it was impossible to confirm the intraventricular location of the electrode by passing the catheter on into the right pulmonary artery. The course of the catheter as determined in posteroanterior projection by fluoroscopy and roentgenography was consistent with the location of the catheter in the right ventricle of these individuals but roentgen-ray observations with the subjects in the right anterior oblique position were not carried out. In the third subject (subject 8, figures 7*A–E* in the paper referred to) the catheter was successfully passed into the pulmonary artery. In these three individuals upright or predominantly upright rapid auricular deflections and prominent ventricular Q waves were recorded. Simultaneous electrocardiograms showed the auricular intrinsic deflection to be inscribed during the latter one-half of the P wave. It seemed clear that these potentials were developed in the left side of the heart and the possibility of accidental catheterization of the left atrium through an interatrial septal defect was suggested but not proved.

It has been pointed out, however,[15] that the anteroposterior projection of the catheter in the coronary sinus may be quite similar to that of the catheter in the right ventricle. The differentiation between these two possible courses of the catheter requires roentgen-ray examination in the right oblique position. Furthermore, the potentials recorded in the three subjects described above are identical with those recorded in the present study when the coronary sinus was deliberately catheterized. Accidental catheterization of the coronary sinus, therefore, seems the most likely explanation for the electrographic findings in at least two (subjects 6 and 26) of the three instances described. The explanation for the findings in the third (subject 8) individual is not clear.

This digression has been deliberately pursued in some detail to emphasize the possibility of confusion in electrographic studies of the human heart by accidental catheterization of the coronary sinus.

Arrhythmias

The problem of arrhythmias during coronary venous catheterization should be evaluated as a possible hazard of the procedure. Nodal and auricular premature beats have been reported in dogs during insertion of the catheter into the coronary venous system, but they have not been reported to occur once the catheter had been left in position.[15] No ventricular premature beats were observed. Similar supraventricular beats are here reported in man, again found only during actual manipulation of the catheter within the coronary sinus or great cardiac vein. In the distal part of the middle cardiac vein, however, ventricular premature beats occurred even after the catheter had been left in place. Myocardial irritation here may have corresponded to the intimate relationship of the middle cardiac vein to the subepicardial myocardium. By contrast, the coronary sinus and proximal part of the great cardiac vein are embedded in a relatively appreciable amount of fatty tissue which may shield the myocardium from contact with the tip of the catheter.

Significant ventricular arrhythmias are commonly noted if the catheter tip is passed beyond the tricuspid valve into the right ventricle, and constitute a conceivable hazard to the usual cardiac catheterization procedures. The absence of anything but transient supraventricular premature beats during catheterization of the coronary sinus and great cardiac vein contributes to the relative safety of this procedure.

Summary and Conclusions

Catheterization of the coronary venous system in 6 individuals with normal hearts appeared to be a relatively safe procedure. Supraventricular premature beats developed during manipulation of the catheter within the great cardiac vein or coronary sinus. Ventricular premature beats were recorded with the catheter in the middle cardiac vein.

As confirmatory evidence of successful coronary venous catheterization, coronary venous blood may be identified grossly by its dark color, and chemically by its reduced content of oxygen, pyruvate, lactate and glucose, when compared with arterial or mixed venous blood. However, as an immediate aid to fluoroscopic and manometric control of the catheterization procedure, electrograms recorded from the catheter tip may serve to determine its location within the chambers of the heart and the various regions of the coronary venous system.

The electrogram in the distal portions of the middle cardiac vein showed a ventricular R wave in one subject, a qR in another; in the proximal portions of this vein there was recorded a ventricular QS in one case, an rS in another. These middle cardiac vein potentials correspond respectively to left and right ventricular epicardial potentials, the inscription of a small initial Q or R wave apparently depending upon the relation of the electrode to the interventricular septum.

The electrical potentials recorded in the coronary sinus and the great cardiac vein of 6 individuals with normal hearts showed a large rapid upward auricular deflection with the intrinsic deflection recorded at the height of or during the latter half of the P wave of simultaneously recorded Lead V_F. This deflection may continue as an Sa wave apparently depending upon whether more atrial muscle is stimulated beyond the position of the recording electrode. The ventricular complex consists of a broad prominent Q wave followed by a prominent R wave and an inverted or biphasic T wave. These tracings probably represent the composite effect of the accessible left atrial and ventricular cavity and epicardial potentials.

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Health, and will be presented in more detail elsewhere.[19]

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QRS-T Patterns in Multiple Precordial Leads That May Be Mistaken for Myocardial Infarction

III. Bundle Branch Block

By GORDON B. MYERS, M.D.

The electrocardiograms of patients in whom myocardial infarction was excluded at autopsy are presented to bring out (1) the following features of left bundle branch block suggesting anteroseptal infarction—QS or qrS patterns in leads from the right precordium or localized to intermediate leads and/or cove plane inversion of the T waves near the transitional zone; (2) the following features of right bundle branch block suggesting infarction—pseudo Q waves, elevated RS-T segments and inverted T waves in right precordial leads, prominent Q waves in leads from the left precordium.

IT IS WELL known that bundle branch block may accompany myocardial infarction or may occur independently. The diagnosis of associated myocardial infarction and bundle branch block has been considered,[1–3] but little attention has been given to the electrocardiographic features of uncomplicated bundle branch block that may be mistaken for those of myocardial infarction. These features will be brought out through the presentation of four cases of left bundle branch block and seven cases of right bundle branch block, in which myocardial infarction was diagnosed or at least considered from the electrocardiographic findings during life, but was subsequently excluded at autopsy. The method of electrocardiographic and pathologic study is similar to that described in a preceding article.[4]

LEFT BUNDLE BRANCH BLOCK

A diagnosis of left bundle branch block is based upon the following criteria[1]: (1) a QRS interval of 0.12 second or longer in complexes derived from impulses of sinoauricular origin and associated with a P-R interval of at least 0.12 second*; (2) an initial upstroke in all leads facing the epicardial surface of the left ventricle, due to early positivity of the left ventricular cavity; (3) a broad, slurred, or notched R wave with abnormally delayed intrinsicoid deflection in the same leads, indicating late arrival of the impulse at the epicardial surface of the left ventricle. The QRS complexes in left ventricular leads are characteristically followed by: depression of the RS-T junction, upward convexity of the RS-T segment, and sharp inversion of the T wave.

The presence of the foregoing QRS-T pattern in V_4, V_5 and V_6, in V_5 and V_6, or even in V_6 alone should lead to a diagnosis of left bundle branch and should not be mistakenly interpreted as due to myocardial infarction. In fact, the diagnostic error likely to be made in the interpretation of left ventricular leads in the presence of left bundle branch block consists in failure to detect coexistent anterolateral infarction.[1,3] The initial R wave produced in left ventricular leads by septal activation from impulses distributed through the right Purkinje network persists when left bundle branch block is complicated by anterolateral infarction and thus prevents the registration of abnormal Q patterns.

In the presence of left bundle branch block, the impulse arrives early in the right side of the septum and free wall of the right ventricle and late in the free wall of the left ventricle. The late activation of the outer wall of the left

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* This stipulation is essential to eliminate broad QRS complexes associated with impulses of ectopic origin or with the Wolff-Parkinson-White syndrome.

ventricle is always manifested by a deep, broad, slurred S wave in leads facing the epicardial surface of the right ventricle and atrium. The initial phase of the QRS complex in these leads depends upon the interplay of negative potentials, transmitted from the endocardial surface of the right side of the septum, and positive potentials produced by activation of the free wall of the right ventricle and perhaps positive potentials developing in the left ventricular cavity and transmitted via the left and right atria to the right precordium. As a consequence, the QRS pattern in leads over the right atrium and ventricle in patients with uncomplicated left bundle branch block may take one of three forms: (1) an rS complex,* consisting of a small, narrow initial r wave due to early preponderance of positive potentials coming from activation of the free wall of the right ventricle and a deep, broad, slurred S wave due to activation of the free wall of the left ventricle; (2) a triphasic qrS complex,[3, 5] the q wave representing initial negativity of the right ventricular cavity from septal activation, the r wave reflecting passage of the impulse through the free wall of the right ventricle; (3) a deep, broad, slurred QS deflection.[6] The r wave may be submerged and obscured in this monophasic downstroke because of complete dominance of negative potentials transmitted from the endocardial surface of the right side of the septum over positive potentials coming from activation of the free wall of the right ventricle, or the r wave may be represented by an equivalent in the form of notching or coarse slurring near the onset of the QS deflection.

Both the QS and the qrS patterns, which may be recorded in leads over the right atrium and ventricle in uncomplicated left bundle branch block, resemble the findings obtained in left ventricular leads as a result of transmural infarction. Furthermore, these QS or qrS deflections are generally followed by abnormal elevation of the RS-T junction and a tall upright T wave. The RS-T segment may exhibit straightening or upward convexity instead of the usual upward concavity, especially when digitalis has been given. The combination of an abnormally elevated RS-T junction and a straightened or convex RS-T segment is strongly suggestive of the RS-T pattern found in the stage of injury associated with infarction of the outer wall of the left ventricle. In the presence of right ventricular dilatation, coupled with clockwise rotation of the heart, the potential variations of the epicardial surface of the right ventricle may be transmitted as far to the left as the anterior axillary line and occasionally even to the midaxillary line. The registration of a broad, slurred QS deflection and an abnormally elevated, convex, or straightened RS-T segment in Leads V_4, V_5, and V_6, which ordinarily reflect the potential variations of the left ventricle, may lead to an erroneous diagnosis of anterolateral infarction unless due consideration is given to the presence of left bundle branch block and the displacement of the transitional zone far to the left.

Another diagnostic error may arise from the fact that leads over the right atrium and ventricle in uncomplicated left bundle branch block do not necessarily show either the expected increase in the initial R wave as the electrode is moved towards the left or the uniform QS or rS pattern occurring as a recognized variant. A distinct rS deflection (or rarely a qrS complex) may be recorded in Lead V_1 or V_2 and a QS deflection in one or more leads farther to the left in the presence of uncomplicated left bundle branch block and may lead to an erroneous diagnosis of anterior infarction. The distinct R wave in Lead V_1 and/or V_2 may be correlated with an electrode position over the tricuspid orifice favorable to the reception of positive potentials coming from the relatively thick base of the right ventricle and perhaps from the left ventricular cavity via the left and right atria, whereas the decrease or disappearance of the R wave in V_3 and V_4 may be correlated with an electrode position over the relatively thin apex of the right ventricle and nearer the right side of the septum, which transmits opposing negative potentials.

An unusual respiratory variation that might raise the question of myocardial infarction was encountered in a case of left bundle branch block reported by Lapin and Sprague.[7] This

* The lower case letter is used to indicate a relatively small deflection, the upper case letter to indicate a relatively large deflection.

was characterized by the registration of a broad, notched QS deflection in transitional lead V_5 during inspiration and a broad, notched R wave in the same lead during expiration. This was attributable to sufficient cardiac rotation in a clockwise direction during inspiration so that the electrode received chiefly the potential variations of the epicardial surface of the right ventricle and sufficient rotation in a counterclockwise direction during expiration so that the record was then dominated by the potential variations of the epicardial surface of the left ventricle. Another extraordinary respiratory variation was observed in Leads V_6 and aV_L of the same patient. This was characterized by the inspiratory registration of an abnormal Q wave followed by a broad, notched R wave, strongly suggestive of the pattern associated with an anterolateral infarct of dense distribution in the subendocardial layer and patchy in the remainder of the wall.[8] However, the inscription of a broad, notched, initial R wave of greater amplitude in the same leads during expiration indicated that the QR pattern was a transitional zonal phenomenon in a case of left bundle branch block. The initial Q wave was probably transmitted from the epicardial surface of the right ventricle and the subsequent R wave was undoubtedly transmitted from the epicardial surface of the left ventricle. This might have occurred as a result of an electrode position that straddled the intraventricular septum, but was more likely due to a shift in cardiac position during the inscription of the QRS as a result of contraction of the right ventricle ahead of the left.

Another pattern occasionally recorded at the transitional zone in left bundle branch block, but likely to be mistaken for that due to myocardial infarction, consists in a QS deflection, accompanied either by elevation of the RS-T junction and inversion of the terminal portion of the T wave or by an isoelectric RS-T segment and deeply inverted T wave. The QS deflection and the elevated RS-T segment resemble those in leads farther to the right and represent predominant transmission of the potential variations of the epicardial surface of the right ventricle during the first part of the cycle. The sharp inversion of the terminal portion of the T or the entire wave resembles that in leads farther to the left and represents predominant transmission of the potential variations of the epicardial surface of the left ventricle during the last part of the cycle. Such a change may be observed as a result of counterclockwise rotation of the heart during left ventricular con-

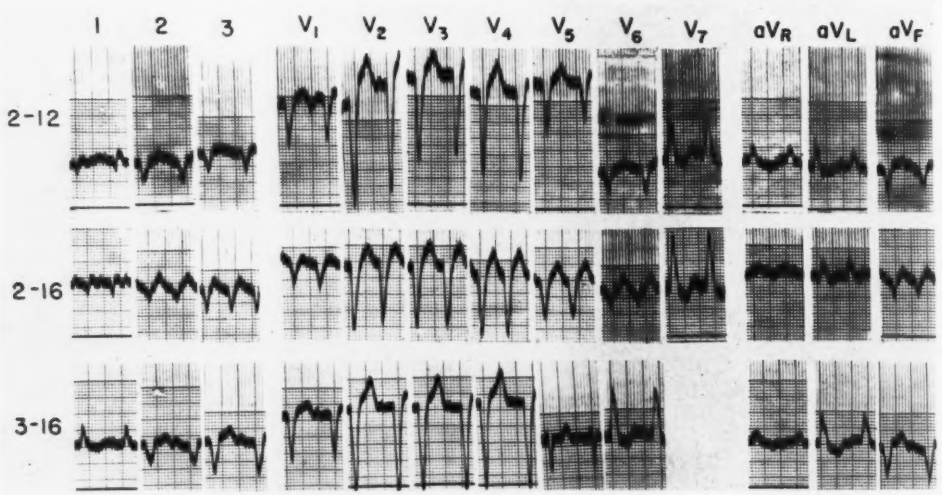

FIG. 1.—Serial electrocardiograms in Case 28.

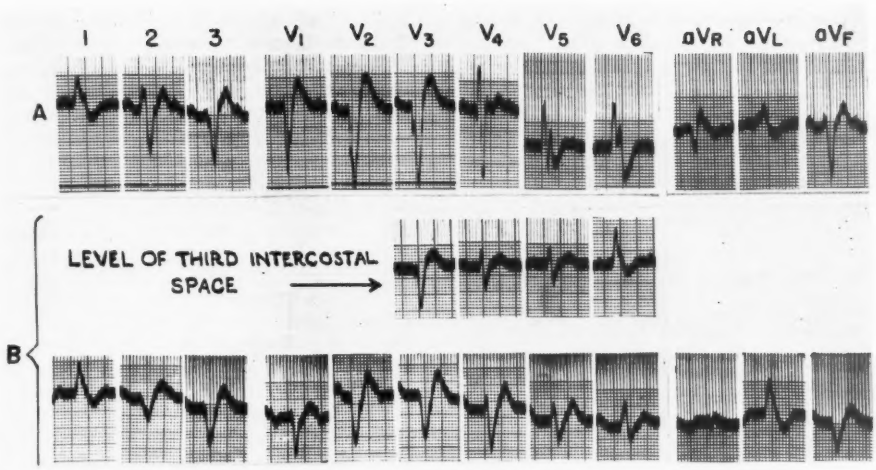

FIG. 2.—Electrocardiographic findings in left bundle branch block. *A*, Case 29; *B*, Case 30.

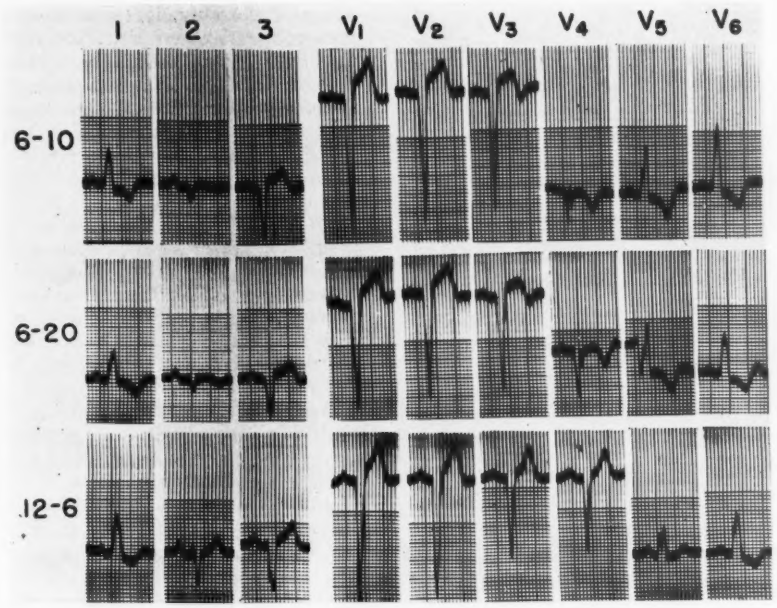

FIG. 3.—Serial electrocardiograms in Case 31.

traction which occurs between the inscription of the QRS and T waves.

A case similar to that of Lapin and Sprague has not as yet been encountered in our autopsy material. The other features of uncomplicated left bundle branch block that may be mistaken for those of myocardial infarction are illustrated in figs. 1, 2, and 3, representing the electrocardiograms in Cases 28 through 31, inclusive.

Broad Slurred QS Deflections in the First Four, Five, or Six Precordial Leads

This may occur in association with left bundle branch block as a result of an associated

dilatation of the right atrium and ventricle and consequent displacement of the transitional zone to the left. The registration of a broad, slurred QS deflection in Leads V_4, V_5, and V_6 may be mistakenly attributed to myocardial infarction, particularly when accompanied by an abnormally elevated, convex RS-T segment. The problem in diagnosis is exemplified by Case 28.

Case 28.—The patient, a man 64 years of age, gave a history of exertional dyspnea for six months and three attacks of sudden weakness, dyspnea, and cyanosis during the three-week period prior to admission to the hospital on Feb. 12, 1946. Physical and roentgen-ray examination revealed evidence of marked enlargement of both the right and left ventricles, with the apical impulse in the midaxilla. There was marked congestion and patchy consolidation of both lung bases, but only moderate peripheral edema. The blood pressure was consistently subnormal.

The tracings reproduced in figure 1 were selected from a series taken during a two-month period of hospitalization. The first two tracings were made on the first and fifth hospital days, when the patient was in congestive failure, and the last was representative of a series taken after compensation was restored. No cardiac glycosides were given because of a clinical diagnosis of recent myocardial infarction.

On February 12, Lead V_1 displayed a QS deflection, followed by an elevated, convex RS-T segment and an apparent cove inversion of the T wave. However, careful inspection showed that the negative phase was due to superimposition of an inverted P wave. Lead V_2 displayed an initial r wave, averaging 1 mm. in amplitude, followed by a broad, deep S wave and an abnormally elevated RS-T junction. The presence of a distinct initial r wave in V_2, together with QS deflections and abnormally elevated RS-T junctions in V_3 and V_4 and QS deflections in V_5 and V_6 led to a diagnosis of a large transmural anterolateral infarct.

A second tracing on February 16 was essentially the same, but showed the following minor alterations, which were taken as evidence supporting the diagnosis of infarction: reduction of the initial r wave in V_2; change from a concave to a convex RS-T segment in V_3 and V_4; the appearance of a late R wave in V_6, resulting in a Qr complex strongly suggestive of the pattern associated with an incomplete transmural infarct. A QS deflection was also recorded in Lead aV_F on both occasions and was incorrectly interpreted as evidence of continuation of an anterolateral infarct into the posterior wall of the left ventricle. Lead I of the second tracing displayed a relatively deep Q wave and a small, but broad, slurred R wave suggestive of lateral infarction, but actually due to greater initial positivity of the right arm than of the left arm.

The inadequacy of the six customary precordial leads for diagnostic purposes was indicated by the lack of contrasting QRS-T patterns in V_1 and V_6 and led to the registration of V_7 on both occasions. A broad, slurred R wave of 0.12 second's duration was recorded in V_7 and was incorrectly interpreted as evidence of delayed conduction through the uninfarcted posterolateral wall of the left ventricle. Upon restudy of these two tracings, it is evident that the findings in V_7 and aV_L were compatible with those recorded over the left ventricle in left bundle branch block and the findings in V_1 through V_6 were in keeping with those recorded over a markedly dilated right ventricle in the absence of infarction. Since the P wave was inverted in V_1 and upright in V_2, the electrode in the latter position was apparently in the vicinity of the tricuspid orifice. The initial r in V_2 could have been derived from activation of a relatively thick ring of muscle at the tricuspid orifice, whereas the initial downstroke in the next four leads could be explained by preponderance of negative potentials transmitted from the right side of the septum over weak positive potentials coming from activation of a dilated, thin-walled apex of the right ventricle. The lower voltage in V_6 than in V_5 or V_7 should have indicated that V_6 was a transitional lead. The deep Q wave in V_6, therefore, could have represented negative potentials transmitted from the epicardial surface of the right ventricle, whereas the late R wave on February 16 could have been derived from activation of the adjoining left ventricle. The QS deflection in aV_F could have been a manifestation of horizontal position in which the potential variations of the epicardial surface of the right ventricle are transmitted to the left leg. Thus, all findings on February 12 and February 16 could be explained by a diagnosis of left bundle branch block with marked right ventricular dilatation, but without infarction.

On March 16, a broad, slurred R wave was inscribed in V_6 similar to that previously recorded in V_7. This change was in keeping with reduction in cardiac size noted on physical and roentgen-ray examination, permitting reference of left ventricular potentials to midaxilla. The initial r wave had disappeared from V_2 and a QS deflection, elevated RS-T junction, and tall, erect T wave were recorded in the first four precordial leads. Lead V_5 displayed a triphasic qrS complex, consisting of a 1-mm. q, 2-mm. r, and 8-mm. S wave, compatible with the pattern sometimes found in left ventricular leads in the presence of patchy transmural infarction or in right ventricular leads in the presence of septal infarction. However, the presence of a broad, slurred R wave in V_6 and aV_L, indicative of left bundle branch block, should have suggested that the pattern in V_5 represented a variant occasionally found in right ventricular or transitional leads. The q wave could have represented negative potentials transmitted from the right side of the septum, the slurred r wave could have been derived from activation of

the adjacent anteroseptal walls of the right and left ventricles, the deep s wave from activation of the lateral and posterior walls of the left ventricle. Hence the findings in the first five precordial leads on March 16 could be explained by right ventricular dilatation in the presence of left bundle branch block, but without infarction.

The patient had further attacks of sudden weakness and dyspnea on April 7 and April 11, the latter ending fatally. Death was due to pulmonary embolism and autopsy revealed multiple, old, organized pulmonary infarcts, which were apparently responsible for the attacks of weakness and dyspnea leading to admission to the hospital. At autopsy the heart weighed 536 grams, which represented marked hypertrophy in view of a body weight of only 40 kilograms. The increased cardiac weight was due chiefly to left ventricular hypertrophy, believed secondary to hypertension because of the presence of advanced renal arteriolosclerosis. There was marked dilatation of the right ventricle, sufficient to explain the displacement of the transitional zone to the left. The coronary vessels were of normal caliber and no evidence of myocardial infarction was found on gross examination or in multiple microscopic blocks. There was patchy fibrosis, which may have been responsible for the conduction defect. In view of the pathologic findings, the abnormalities in the electrocardiogram were evidently the result of left bundle branch block complicated by marked dilatation of the right ventricle.

Progressive Diminution of the Initial R Wave, Then Replacement by a qRS Complex as the Electrode is Moved Towards the Left

These findings arouse the suspicion of myocardial infarction, but may occur in association with uncomplicated left bundle branch block, as exemplified by Case 29.

Case 29.—The patient, a man, age 73 years, showed evidence of congestive failure secondary to hypertension and uremia associated with prostatic obstruction.

The electrocardiogram reproduced in Fig. 2*A* was obtained after digitalization and approximately twelve hours before death. A diagnosis of left bundle branch block was easily established by the QRS interval of 0.14 second together with the broad, notched initial R wave in Leads V_5 and V_6. The extreme depression of the RS-T junction in these leads and the abrupt return of the string to the isoelectric line to complete the T wave were extraordinary and raised the question of subendocardial ischemia. However, the short Q-T interval and the contour of the ascending limb of the T wave indicated that overdigitalization was largely, if not entirely, responsible.

The first three precordial leads displayed an rS deflection and Lead V_4 showed a qRS complex. The suspicion of myocardial infarction was aroused by: (1) the decrease in the initial r wave from 2.0 mm. in V_1 to 1.0 mm. in V_2 and to 0.5 mm. in V_3; (2) the replacement of the initial r by a 2.5-mm. q wave in V_4; (3) the marked elevation and domelike contour of the RS-T segments in the first three precordial leads. The deep, broad S wave in V_1, V_2 and V_3 indicated that the electrode was over the right side of the heart, and the marked upward displacement and rounding of the RS-T segments in these leads could have been produced by digitalis effects superimposed upon those of uncomplicated left bundle branch block. The relatively large initial R wave in V_1 could be accounted for by placement of the electrode over the right atrium in a position to receive positive potentials produced by activation of the relatively thick base of the right ventricle. The progressive reduction in amplitude of the initial r wave in the first three precordial leads could be explained by shift of the electrode from the vicinity of the tricuspid ring to points over the thin apex of the right ventricle, where it was in a position to receive relatively weak positive potentials coming from activation of the free wall of the right ventricle and simultaneous negative potentials transmitted from the endocardial surface of the right side of the septum. The qRS pattern in V_4 was in keeping with an electrode position that straddled the septum. The small q could have represented normal initial negativity transmitted from the subjacent endocardial surface of the right side of the septum; the timing and amplitude of the succeeding R wave indicated transmission from the adjoining anteroseptal wall of the left ventricle and the final S wave was derived from activation of lateral and posterior walls of the left ventricle. Thus, all atypical features of this electrocardiogram could be explained without postulating complicating infarction.

At autopsy the heart weighed 700 grams and showed marked left ventricular hypertrophy, but no evidence of myocardial infarction. The coronary vessels were of normal caliber and showed only mild sclerosis.

An rS Deflection in Lead V_1 and/or V_2 and a QS in Leads Farther to the Left

This may occur in association with uncomplicated left bundle branch block and may lead to an erroneous diagnosis of myocardial infarction. This finding was encountered in the first two tracings of Case 28, discussed above, and will be exemplified further by Case 30.

Case 30.—The patient was a man, age 66 years, who had been incapacitated because of congestive failure for five months, in spite of the administration of digitoxin and diuretics by his family physician. He was admitted to the hospital with congestive failure complicated by shock associated with unobtainable blood pressure.

The routine electrocardiogram reproduced in figure 2*B* was obtained on the first hospital day and the additional high precordial leads were taken three days later. Digitoxin was continued in doses of 0.2 mg. daily throughout this period. The P-R interval was lengthened to 0.24 second and the QRS interval to 0.16 second. The presence of left bundle branch block was indicated by the broad, slurred initial R waves in left ventricular leads V_6 and aV_L. An upright T wave was recorded in V_6, instead of the inverted T wave usually found in left ventricular leads in the presence of left bundle branch block. Concordant T waves represent an occasional finding in uncomplicated bundle branch block and do not, in themselves, constitute evidence of myocardial infarction.

The atypical features of the tracing that raised the question of infarction consisted in the registration of a distinct initial r wave in V_1 and a QS complex with abnormally elevated, convex RS-T segments in V_2 and V_3. The diphasic P wave in V_1 indicated that the electrode was over the right atrium, whereas the upright P waves in V_2 and V_3 suggested that these positions were over the right ventricle. The initial R wave in V_1 was presumably derived from activation of the relatively thick ring of muscle at the tricuspid orifice. The disappearance of the R wave in V_2 and V_3 as the electrode approached the septum could be explained by preponderance of negative potentials transmitted from the right side of the septum over weaker positive potentials produced by activation of the thinner apical portion of the right ventricle.

High precordial leads were taken at the intersections of a horizontal line through the junction of third intercostal space and sternum with vertical lines through the V_3, V_4, V_5, and V_6 positions. The records taken high in the anterior axillary and midaxillary lines resembled those taken in the customary V_5 and V_6 positions, respectively. The electrode in both the high and the standard V_3 and V_4 positions was apparently to the right of the septum, as judged by the relatively deep and broad S waves. The tracing in the high V_3 position displayed a triphasic qrS complex, consisting of a q wave 1.5 mm. deep, an r wave that barely crossed the isoelectric line and an S wave 11 mm. deep, followed by an abnormally elevated, dome-shaped RS-T segment. The contour of both the qrS and the RS-T complexes in high V_3 was strongly suggestive of infarction. However, in view of the left bundle branch block a qrS of this type could be recorded in a right ventricular lead in the absence of infarction. The q wave reflected initial negativity of the right ventricular cavity, as a result of reversal in the vector associated with septal activation; the r wave was produced by passage of the impulse through the free wall of the right ventricle. The elevated domelike RS-T segment was the reciprocal of the depressed V-shaped segment in left ventricular Lead aV_L and was attributable to digitalis effect. Hence the findings in both the customary and high precordial leads could be explained by uncomplicated left bundle branch block.

The patient died in congestive failure on the fifth hospital day. At autopsy the heart weighed 607 grams and showed marked left ventricular hypertrophy of hypertensive origin, and secondary right ventricular dilatation. The coronary vessels were of normal caliber and revealed only mild atherosclerosis. No evidence of myocardial infarction was found on gross examination or in multiple microscopic blocks.

Triphasic qrS Pattern in One or More Precordial Leads Just to the Right of the Transitional Zone

A triphasic qrS complex, consisting of small, narrow q and r deflections and a deep, broad S wave, is occasionally recorded in right ventricular leads in association with uncomplicated left bundle branch block, as illustrated by Lead V_5 of the last tracing in Case 28 and by high Leads V_3 and V_4 of Case 30. The origin and significance of this pattern have been discussed above and also in connection with the report of Case 99 of a previous manuscript.[5]

QS or W-Shaped Complexes Together With Cove-Plane Inversion of the T Waves in Intermediate Leads Near the Transitional Zone

Findings of this type in Leads V_3 and V_4 of Case 31, figure 3, led to an electrocardiographic diagnosis of anteroseptal infarction that could not be substantiated at autopsy.

Case 31.—The patient, a housewife, age 52 years, was known to have had diabetes and hypertension for one year. On June 3, 1944, she was awakened from a sound sleep by a severe choking sensation with accompanying orthopnea, which lasted for approximately one hour. Paroxysmal dyspnea recurred on the two succeeding nights and was not accompanied by pain. She was admitted to the hospital on June 6 with marked pulmonary edema and slight peripheral edema, and received a total of 1.0 Gm. digitalis leaf up until June 13, at which time the drug was discontinued. Compensation was rapidly restored and the hospital course was uneventful. There was no fever, leukocytosis, or elevation in sedimentation rate.

The electrocardiograms reproduced in figure 3 were selected from a series taken over a period of six months. The QRS-T patterns in five electrocardiograms taken after August 1 corresponded closely and consequently only the final tracing, on December 6, is included in the illustration. Left

bundle branch block was present throughout, as indicated by (1) a QRS duration of 0.12 second or more, (2) a broad, slurred initial R wave in Leads V_5 and V_6. A deep, broad QS deflection, elevated RS-T segment, and tall upright T wave were consistently recorded in the first two precordial leads. A similar QS deflection was also present in Lead V_3 and was accompanied by an elevated domelike RS-T segment and sharply inverted T wave in June, and by a normal RS-T complex after the first of August. The tracings taken at position V_4 in June revealed (1) a triphasic Qrs complex, consisting of a deep Q wave, a small late r wave, which sometimes failed to cross the isoelectric line, and a relatively small S wave; (2) an isoelectric RS-T segment and sharply inverted T wave. In later tracings, Lead V_4 displayed either a minute r and deep broad S wave or a QS deflection like that at V_3, accompanied by an upright T wave. On the basis of the QRS-T pattern in Leads V_3 and V_4 in June, a diagnosis of recent anteroseptal infarction was made. The change in RS-T complexes during the ten-day interval between the first two tracings was not as great as would be expected in the presence of recent infarction, but the subsequent change in QRS pattern in V_3, along with the evolution to a normal upright T wave in V_3 and V_4, tended to overcome this objection and to support the diagnosis of infarction.

The patient remained under close medical supervision. Digitalization was carried out in July and was maintained for the rest of her life. She had no further attacks of paroxysmal dyspnea and no chest pain. She was readmitted to the hospital on Oct. 23, 1944, with increasing nervousness, heat intolerance, and weight loss of one month's duration. The basal metabolism rate ranged from +38 to +41 and thiouracil was instituted. A satisfactory clinical response was obtained until Jan. 17, 1945, when the white blood count dropped to 3950 per cu. mm. of blood. Thiouracil was stopped, but the white count continued to fall and the patient died of agranulocytosis ten days later.

At autopsy the heart weighed 347 grams and showed slight left ventricular hypertrophy. The coronary tree injected well and the vessels were of normal caliber and showed only minimal atherosclerosis. Meticulous gross examination revealed no evidence of myocardial infarction. There was a small patch of healed epicarditis at the left apex, occupying an area 2.5 cm. in diameter. This lesion was well healed and did not extend into the myocardium either on gross or microscopic examination. Many microscopic blocks were studied, but no evidence of infarction was found. The possibility that anteroapical infarction had occurred in June 1944 and had healed completely except for an epicardial scar could not be ruled out, but was deemed unlikely because of the normal histologic appearance of the myocardium.

Upon restudy of the electrocardiograms, it would appear that the abnormalities in V_3 and V_4, which had been interpreted as evidence of anteroseptal infarction, could have been the result of admixture of right and left ventricular potentials at the transitional zone. The deep QS deflections, elevated RS-T segments, and upright T waves in V_1 and V_2 were representative of the pattern sometimes recorded over the normal right ventricle in the presence of left bundle branch block. The resemblance of the QS deflection and elevated RS-T segment in V_3 to those in V_1 and V_2 suggested that the electrode lay to the right of the septum during the inscription of this portion of the ventricular complex, whereas the resemblance of the inverted terminus of the T wave to that in leads farther to the left suggested that the heart may have rotated sufficiently during the intervening mechanical systole to bring the left ventricle beneath the electrode.

The Qrs pattern in V_4 could also be explained as a manifestation of the transitional zone associated with an electrode position straddling the septum. The Q wave corresponded to the first portion of the QS in the first three precordial leads and was probably transmitted through the anteroseptal wall of the right ventricle. The R wave corresponded to the expected time of activation of the anteroseptal wall of the left ventricle and was probably derived in this manner. The T wave simulated those in left ventricular Leads V_5 and V_6. In left bundle branch block, contraction of the right ventricle may begin before completion of activation of the left and might cause cardiac rotation during inscription of the QRS complex. This may constitute the mechanism for the predominant transmission of the first portion of the QRS from the right ventricle and the last portion from the left ventricle in a lead overlying the septum.

The QRS-T patterns in V_3 and V_4 could therefore have represented a transitional zonal effect in left bundle branch block uncomplicated by myocardial infarction, ischemia, or pericarditis. Whether or not the epicarditis found at autopsy developed in June and contributed to the T-wave inversion is a matter for conjecture. The fact that the T waves in V_3 and V_4 subsequently became upright does not necessarily indicate that either epicarditis or ischemia was responsible for the inversion. The change to upright T waves in V_3 and V_4 was believed secondary to the reversal in the T wave in left ventricular lead V_5. Discordant T waves are occasionally replaced by concordant T waves for no discoverable reason in uncomplicated left bundle branch block.

Right Bundle Branch Block

Uncomplicated right bundle branch block is manifested by[1, 6, 9]: (1) a QRS interval of 0.12 second or longer in complexes derived from an impulse of sinoauricular origin and associated

with a P-R interval of at least 0.12 second; (2) an initial upstroke in all leads facing the epicardial surface of the right ventricle, due to early positivity of the right ventricular cavity; (3) a late secondary upstroke in the same leads, due to delayed activation of the free wall of the right ventricle. The intervening space between these two R waves may be taken up by coarse slurring or by a downstroke that may stop above the isoelectric line to form a notch or carry below to form an S wave. This downstroke reflects negative potentials transmitted to the right precordium during the brief interim between activation of the septum and the free wall of the right ventricle. The QRS complexes in right ventricular leads are characteristically followed by: depression of the RS-T junction, upward convexity of the RS-T segment, and sharp inversion of the T wave.

Right bundle branch block secondary to septal infarction is distinguished from the uncomplicated variety by the direction of the initial phase of the QRS complex in leads from the right precordium.[1–3] An abnormal Q wave is characteristically found in these leads in the presence of septal infarction, whereas an initial upstroke should be detectable in uncomplicated right bundle branch block. When recent septal infarction is responsible for the conduction defect, right precordial leads generally show elevation of the RS-T junction and cove inversion of the T wave, in contrast to the RS-T depression usually associated with uncomplicated right bundle branch block.

The following variants may occur in the pattern of uncomplicated right bundle branch block and may lead to an erroneous diagnosis of septal infarction: (1) pseudo Q waves in leads from the right precordium and/or transitional zone; (2) elevation of the RS-T junction and abnormal inversion of the T wave in leads from the right precordium; (3) reduction in the R wave and deep inversion of the T wave in intermediate leads; (4) concordant T waves; (5) prominent Q waves in leads from the left precordium. These features are collectively illustrated by the electrocardiograms in figure 4, obtained in Cases 32 to 38, inclusive.

Pseudo Q Waves in Leads from the Right Precordium and/or Transitional Zone

The initial R wave transmitted to the right precordium as the result of septal activation in association with uncomplicated right bundle branch block progressively diminishes with increasing distance from the septum and may be barely detectable in leads over or beyond the right atrium. If a minute initial R wave is overlooked, the succeeding downstroke may be misinterpreted as a Q wave and an erroneous diagnosis of septal infarction may be made. The problem is exemplified by Cases 32 and 33. The electrocardiograms are reproduced in figures 4*A* and *B*.

Case 32.—The patient, a man, age 51 years, had had recurrent asthmatic bronchitis over a period of four years and was admitted to the hospital with left-sided bronchopneumonia and pleurisy. The electrocardiogram reproduced in figure 4*A* was obtained during the febrile period, after partial digitalization. At first glance, there appeared to be an inital downstroke and late R wave in V_1 and V_2 and a deep, notched QS complex in V_3. If this had constituted a correct identification of the components of the QRS complex, a diagnosis of septal infarction would have been justifiable. However, close scrutiny revealed a small but distinct initial r wave, averaging 0.5 mm. in amplitude, in each of the three leads. This initial r wave was a manifestation of septal activation and the succeeding downstroke was therefore an S wave, commonly found in association with uncomplicated right bundle branch block.

In searching for an explanation for the exceptionally small initial r wave recorded in right precordial leads V_1 and V_2, an unusually high position of the electrode was suggested by the following observations: (1) emphysematous depression of the diaphragms, which tended to lower the heart in reference to fixed points on the thoracic cage; (2) inverted P waves in the presence of sinus rhythm, recorded not only in right ventricular leads V_1 and V_2, but also in transitional lead V_3 and in left ventricular lead V_4. The configuration of the P waves suggested that the electrode lay over the atria and was therefore in a position to receive potential variations of the two ventricular cavities. Simultaneous transmission of negative potentials through the mitral orifice to supraventricular positions V_1, V_2 and V_3 nearly succeeded in neutralizing positive potentials coming from the right ventricular cavity, thereby accounting for the unusually small initial r wave.

A noteworthy feature of the last four precordial leads was the 1- to 3-mm. elevation of the RS-T

junctions and the straightening of the RS-T segments. Although digitalis tends to straighten the RS-T segments and displace them in a direction opposite to the main deflection of the QRS complex, it was not responsible for the RS-T abnormalities in this case because of: (1) absence of Q-T shortening; (2) presence of a similar degree of RS-T segment elevation in a tracing made one week previously, before the administration of glycosides. The contour of the elevated RS-T segments in V_4, V_5, and V_6 was strongly suggestive of a lesion of the subepicardial layer of myocardium and aroused the suspicion of infarction and pericarditis. The possibility of transmural infarction could be virtually excluded by the absence of Q waves, but the question remained as to whether the subepicardial lesion was the result of superficial infarction or pericarditis.

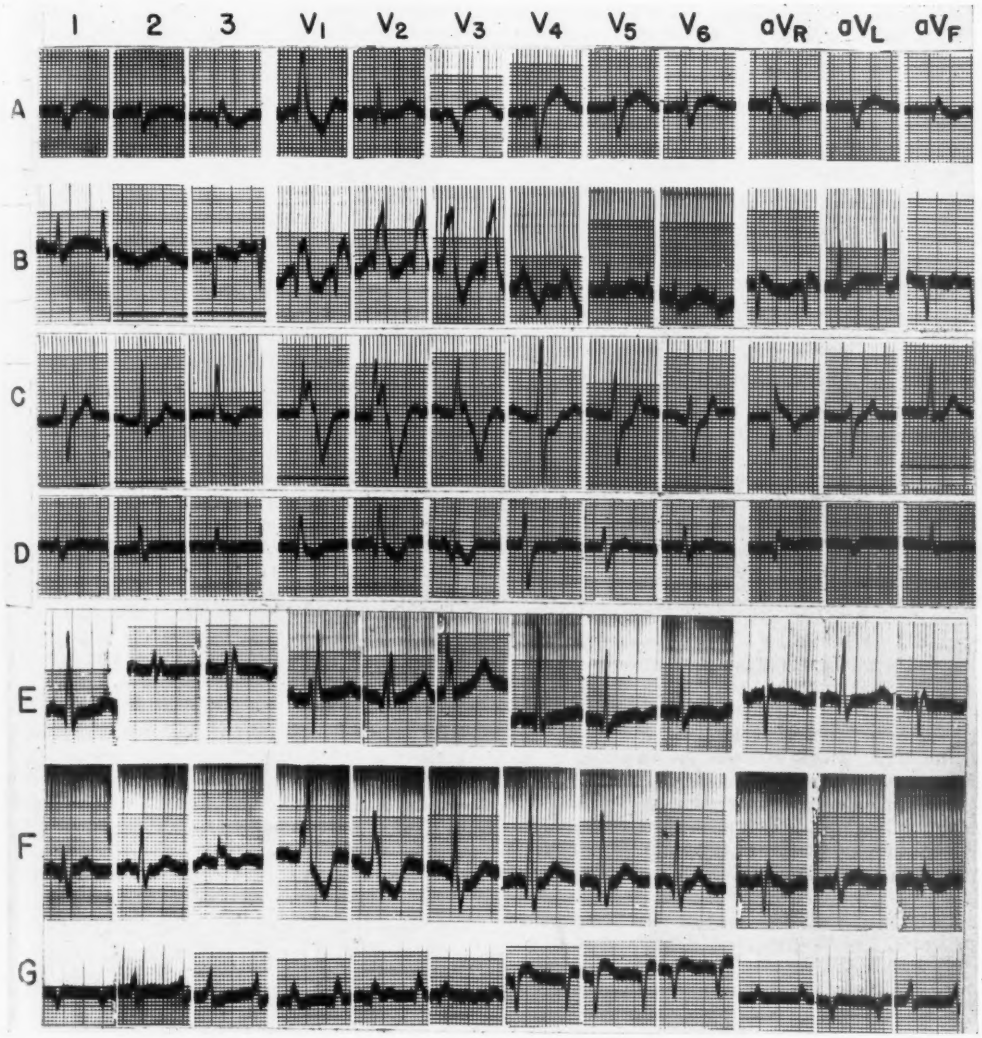

FIG. 4.—Electrocardiographic findings in right bundle branch block. *A*, Case 32; *B*, Case 33; *C*, Case 34; *D*, Case 35; *E*, Case 36; *F*, Case 37; *G*, Case 38.

Death occurred five days after the electrocardiogram was made. At autopsy the heart weighed 387 grams and showed moderate right ventricular hyper-

trophy secondary to chronic obstructive pulmonary emphysema. There was no evidence of coronary narrowing or myocardial infarction. No lesion other than right ventricular hypertrophy was found to account for the right bundle branch block, but the possibility of microscopic degeneration or fibrosis in the right branch of the bundle of His was not excluded, because of failure to take serial microscopic sections in this area. A subacute epicarditis was found over the anterolateral aspect of the left ventricle and extended sufficiently into the subepicardial layer of myocardium to explain the RS-T elevation in V_4, V_5 and V_6.

Elevation of the RS-T Junction and/or Abnormal Inversion of the T Wave in Leads from the Right Precordium

RS-T patterns of this type in association with right bundle branch block are strongly suggestive of septal infarction, but may occur independently, as exemplified by Cases 33 and 34. Electrocardiograms in these cases are reproduced in figs. 4*B* and *C*.

Case 33.—The patient, a man, 74 years of age, suffered acute left ventricular failure following a ureterosigmoid transplant necessitated by carcinoma of the bladder. The electrocardiogram reproduced in figure 4*B* was obtained after the intravenous administration of 0.8 mg. Cedilanid.

The rhythm was regular throughout, but the rate increased from 130 during the inscription of the limb leads to 150 during the registration of the precordial leads. This observation, together with the widened QRS complexes in the first four precordial leads and the apparent normal duration of the QRS in the remaining leads, aroused the suspicion of a transient episode of ventricular tachycardia, beginning between the registration of Leads aV_F and V_1 and terminating between the inscription of Leads V_4 and V_5. From a more careful study of the tracing, the following evidence was elicited, indicating that a sinus tachycardia was present throughout: (1) distinct upright P waves were found in V_1 and V_2, occupying a constant position in reference to the T waves and preceding the QRS complex by the same P-R interval as in the limb leads; (2) the cardiac rate and Q-T interval were the same in V_3 and V_4, where the QRS complexes were widened, as in V_5 and V_6, where the QRS complexes appeared to be of normal duration. The discrepancy in apparent QRS interval was thus due to the fact that the last portion of the QRS was isoelectric or nearly isoelectric in V_5, V_6, and the limb leads. Measurements with the dividers indicated that the depressed segment following the R wave in Leads V_6, aV_L and I and the elevated segment following the downstroke in Leads aV_R, aV_F and III were part of the QRS complex, and that the RS-T segment in these leads was isoelectric and fused with the QRS complex.

From the general resemblance of the QRS complexes in the first four precordial leads it was concluded that they represented predominantly the potential variations of the right side of the septum and epicardial surface of the right ventricle. The broad R wave and late intrinsicoid deflection in these leads, together with the broad S wave in left ventricular leads V_6 and aV_L, indicated that the sinus tachycardia was complicated by right bundle branch block.

Unless careful inspection were made of the QRS complexes in the first three precordial leads, the downstroke might be misinterpreted as a Q wave and mistaken as evidence of myocardial infarction. However, close scrutiny revealed a small but distinct initial R wave in each lead. Still more confusing, however, was the apparent RS-T displacement in V_1 and V_2. It might seem, from first glance at V_1, that the RS-T junction was located at the peak of the upstroke, 6 mm. above the isoelectric line. In that event, the precipitous descent to an inverted T wave would be strongly suggestive of recent septal infarction. However, the contour of the tracing in this precipitous descent of the string was more in keeping with a portion of the QRS rather than a portion of the RS-T complex. This led to an attempt to ascertain the termination of the QRS complex in V_1 by interpolation of measurements of the QRS interval made in V_3, where the beginning and end of the complex were quite distinct. On the assumption of a similar QRS interval in V_1 and V_2 to that in V_3, the RS-T junction in both leads was located at or slightly below the isoelectric line. Thus, from careful study, it was concluded that both the QRS and RS-T patterns could be accounted for on a basis of uncomplicated right bundle branch block.

Death occurred sixteen hours after the electrocardiogram was made. At autopsy the heart weighed 360 grams and showed a normal ventricular ratio. There was moderate coronary sclerosis, but no narrowing or occlusion. There was no evidence of myocardial infarction. The right bundle branch block was believed secondary to the acute left ventricular failure, but serial sections of the bundle of His were not made to exclude the possibility of a localized lesion.

Another type of RS-T pattern that might be mistaken for that of myocardial infarction is illustrated in figure 4*C*, which represents the electrocardiogram in Case 34.

Case 34.—The patient was an 18 year old girl with chronic glomerulonephritis. The electrocardiogram was obtained shortly before death, which was due to uremia. Blood potassium was not deter-

mined, but was probably elevated in view of marked oliguria and a creatinine level of 18.6 mg. per cent.

A diagnosis of right bundle branch block was readily established from the broad, double-peaked R wave in Leads V_1 and V_2. The possibility of recent septal infarction was suggested by the elevated RS-T junctions and sharply inverted T waves in V_1 and aV_R, and the possibility of continuation of the lesion into the subendocardial layer of the anterolateral wall of the left ventricle was suggested by the RS-T depression in Leads V_4, V_5 and V_6. The absence of Q waves militated against infarction and necessitated search for another explanation for the RS-T pattern. The narrow base and sharp peak of the T waves in V_5 and V_6 were strongly suggestive of hyperpotassemia and the exceptionally deep and sharply inverted T waves in the first three precordial leads could also be explained on the same basis.

At autopsy the heart weighed 350 grams. Left ventricular hypertrophy was present, but gross and microscopic examination revealed no other abnormality.

Reduction of the R Wave and Deep Inversion of the T Wave in Intermediate Leads

Patterns of this type may arouse the suspicion of anteroseptal infarction, but may occur as a transitional zonal phenomenon in association with uncomplicated right bundle branch block, as exemplified by Case 35.

Case 35.—The patient, a woman, 58 years of age, was admitted to the hospital with a typical history of hyperthyroidism of three years' duration, complicated by congestive failure of three weeks' duration. The electrocardiogram reproduced in figure 4*D* was obtained on the fourth hospital day after digitalization. Fine auricular fibrillation was present.

Lead V_2 displayed an rsR′ complex typical of right bundle branch block, followed by the expected RS-T depression and inversion of the T wave. A glance at V_1 would disclose a comparable late R wave and RS-T complex, but the minute initial r might escape detection and thus lead to the misinterpretation of the downstroke as a Q wave and an erroneous diagnosis of septal infarction. Careful scrutiny of a number of cycles revealed a small but distinct initial r wave, ranging from 0.25 to 0 5 mm. in amplitude. The smaller initial r wave in V_1 than in V_2 was presumably due to the fact that the electrode was farther from the right side of the septum, but closer to the right atrium, where it might be in a position to receive negative left ventricular cavity potentials transmitted through the left atrium.

A more confusing pattern was recorded in Lead V_3. At first glance, the QRS might be classed as a W-shaped complex and thus be mistaken as evidence of infarction. However, measurements of the QRS interval indicated that the preliminary oscillations were part of the complex, constituting an initial R, preceding a broad, notched S wave. The fact that the R wave in V_3 was much smaller than the R′ in V_2 and the R wave in V_4 could be ascribed to the transitional zone, but the fact that the inverted T wave in V_3 was significantly deeper and different in shape from that in V_2 and V_4 was not so readily explained. The possibility of anteroseptal infarction was originally entertained from the RS-T pattern in V_3, but was later discarded after subsequent tracings, taken from slightly different points, showed that the inverted T wave was also a localized phenomenon, referable to the transitional zone.

Death occurred three weeks later from intercurrent pneumonia. At autopsy the heart weighed 440 grams. Left ventricular hypertrophy was found and was attributed to thyrotoxicosis after exclusion of other recognized causes. The coronary arteries were of normal caliber and no evidence of myocardial infarction was found. The septum appeared normal on gross and routine microscopic examination, but serial sections in the vicinity of the bundle of His were not made.

Concordant T Waves

Since it is generally recognized that concordant T waves may occur in association with uncomplicated bundle branch block, Case 36 is included more because of the clinical than the electrocardiographic problem that was presented.

Case 36.—The patient, a hypertensive man 65 years of age, was suddenly seized with severe retrosternal constriction and dyspnea and was brought to the hospital in shock. The blood pressure was 60/40 in the left arm and the pulse rate was 50 in the left wrist; neither blood pressure nor pulse rate was obtainable on the right. No cardiac glycosides were given.

The electrocardiogram obtained eight hours after admission is reproduced in figure 4*E*. A sinus arrhythmia was present with cycle lengths ranging from 1.0 to 1.24 seconds. The QRS interval of 0.13 second and the double peaking of the R waves in right ventricular leads V_1, V_2 and V_3 were indicative of right bundle branch block. As the electrode was moved from the V_1 position near the relatively thick base of the right ventricle to the V_3 position over the relatively thin apex of the right ventricle and close to the septum, the initial R wave showed the progressive increase in amplitude expected of a deflection of septal derivation, whereas the R′ wave showed a concomitant decrease, commensurate with the diminishing mass of the free wall of the right ventricle. The progressive decrease in the intervening downstroke was also in keeping with its origin from negative left ventricular cavity po-

tentials, transmitted through the left and right atrium to the right precordium. The Q wave recorded in left ventricular leads V_4, V_5 and V_6 was considered normal, both as to duration and relative amplitude. The slight delay in onset of the intrinsicoid deflection in V_4 and V_5 and the relatively tall R waves in these leads, in the face of right bundle branch block, suggested left ventricular hypertrophy. The Q-T interval of 0.52 second was abnormally long, even for the slow rate.

The T wave was upright and concordant with the double-peaked R wave in the first three precordial leads and, like the initial R wave of septal origin, showed a progressive increase in amplitude as the electrode was moved from the V_1 to the V_3 position. Tall, erect T waves of this configuration in V_2 and V_3 arouse the suspicion of posterolateral infarction, but no evidence of such was found in Lead aV_F or V_6. The inverted T waves in V_5 and V_6 were concordant with the broad, slurred portion of the QRS complex. In summary, the electrocardiogram showed evidence of right bundle branch block and probable left ventricular hypertrophy. The only atypical feature was the concordance of the T waves and this was compatible with uncomplicated bundle branch block. Thus, the electrocardiogram was abnormal, but failed to confirm the suspicion of myocardial infarction engendered by the clinical history.

Death occurred eighteen hours after the electrocardiogram was made and autopsy revealed a dissecting aneurysm of the aorta. The heart weighed 548 grams and showed left ventricular hypertrophy. There was moderate coronary sclerosis, but no narrowing or occlusion and no evidence of myocardial infarction. Thus, a correctly interpreted electrocardiogram in this case furnished valuable negative evidence against myocardial infarction and pointed towards some other cause for the acute retrosternal constriction.

Q Waves in Leads from the Left Precordium

These are not uncommon in association with right bundle branch block, in the absence of myocardial infarction. The duration of these Q waves and the relative amplitude in reference to the succeeding R waves may be sufficiently great, especially in the presence of left ventricular hypertrophy, to raise the question of subendocardial anterolateral infarction. The problem is exemplified by Cases 37 and 38. The electrocardiograms are reproduced in figures 4*F* and *G*.

Case 37.—The patient, a man 67 years of age, had had increasing dyspnea over a period of one year and was admitted to the hospital with severe orthopnea with extensive peripheral edema. There were physical signs of aneurysm of the ascending aorta, complicated by atelectasis of the right upper lobe. Digitalization had been carried out prior to the recording of electrocardiographic tracings.

The presence of right bundle branch block was established by the QRS interval of 0.12 second together with the broad double-peaked R waves in right ventricular leads V_1 and V_2. The excessive RS-T depression and straightening of the RS-T segments in these leads were attributable to superimposed digitalis effects. The transitional zone was situated between the V_2 and the V_3 positions and the potential variations of the epicardial surface of the anterolateral wall of the left ventricle had the predominant effect on the QRS-T pattern in the last four precordial leads. The slight delay in onset of the intrinsicoid deflection in these leads together with the relatively tall R waves, in the face of right bundle branch block, were interpreted as evidence of left ventricular hypertrophy. The presence of Q waves 4 mm. in depth and 25 per cent of the succeeding R waves in V_5 and V_6 raised the question of infarction of the subendocardial layer of the anterolateral wall and the Q/R ratio of 25 per cent in aV_F suggested the possibility of extension into the subendocardial portion of the posterior wall. Inasmuch as the time interval from onset to nadir of the Q waves did not exceed 0.02 second in any lead, and in view of the fact that the Q/R ratios were borderline, but not definitely abnormal, it was concluded that the Q waves in all leads could be accounted for by left ventricular dilatation and hypertrophy in the absence of infarction.

On the day after the electrocardiographic tracings were made, death occurred due to massive pulmonary hemorrhage. Autopsy revealed a large aneurysm that obstructed and eroded the right stem bronchus and compressed the pulmonary artery. The heart weighed 448 grams and showed moderate left ventricular hypertrophy. There was no evidence of coronary narrowing or myocardial infarction.

Case 38.—The patient was a disoriented man, 52 years of age, who was brought to the hospital at midnight with extreme orthopnea and cyanosis, but only moderate peripheral edema. He had had a chronic cough for several years and had been treated for recurrent congestive failure by his family physician during the preceding six months. The chest was barrel-shaped, but auscultation revealed crepitant râles throughout both lung fields. These râles were interpreted as evidence of acute pulmonary edema due to left ventricular failure. Despite the lack of definite information as to the amount of digitalis taken prior to admission, Cedilanid was given intravenously in three divided doses during the night to a total of 1.6 milligrams.

The electrocardiogram reproduced in figure 4*G* was obtained one-half hour before death. The cardiac rhythm appeared regular to auscultation

both before and after the administration of Cedilanid, but the electrocardiogram revealed auricular fibrillation with slightly irregular ventricular rhythm, cycle lengths varying from 0.40 second to 0.52 second. The ventricular complexes were uniform in contour in any given lead. The electrocardiogram was therefore interpreted on the assumption that the impulses responsible for the QRS-T complexes reached the ventricles through the A-V node.

Close scrutiny of Leads V_1 and V_2 revealed a minute initial R wave, a small downstroke, and a prominent slurred R′ deflection, which attained a peak 0.08 second after the onset of the QRS complex. These findings together with a QRS interval of 0.12 second led to a diagnosis of right bundle branch block. The striking features of this case were the qrS complexes and abnormally elevated, domelike RS-T segments in V_4 and V_5 and the QS deflection and rounded RS-T segment in V_6 and aV_L. These findings were attributed to recent infarction of the anterolateral wall of the left ventricle.

Autopsy revealed far-advanced obstructive emphysema and pulmonary arteriosclerosis, with a complicating acute suppurative bronchiolitis to account for the diffuse crepitant râles. The heart weighed 421 grams and showed evidence of marked dilatation and hypertrophy of the right ventricle. The hypertrophy was so marked that the weight of the right ventricular segment exceeded that of the left, as indicated by a ratio of 0.74. The coronary tree was of normal caliber. No evidence of myocardial infarction was found on gross examination or in multiple microscopic blocks taken around the circumference of the ventricle at different levels.

After the necropsy was completed, reconsideration was given to the possibility that a ventricular tachycardia, arising from a focus in the left apex, might have been responsible for the bizarre QRS-T pattern. A QS- or W-shaped complex may be recorded through a lead over the site of origin of the ectopic impulse, but would not be expected over so wide an area as that covered by the V_4, V_5, and V_6 positions. Furthermore, the first portion of the QRS in V_1 and V_2 (i.e., the rs deflection) was atypical of the pattern found in right ventricular leads in ectopic beats arising in the left ventricle. Hence restudy of the tracing affirmed the previous conclusion that the impulses responsible for the QRS-T complexes reached the ventricle by way of the A-V node.

Activation of the free wall of the markedly hypertrophied right ventricle was believed responsible for the prominent R′ wave in V_1 and V_2 and for the deep S wave in V_4, and V_5. Predominant activation of the septum by impulses spreading from left to right apparently led to the inscription of the initial R wave in V_1 and V_2 and to the Q wave in V_4 and V_5. Since the initial r wave in V_1 and V_2 was normal in duration and very low in voltage, whereas the final R′ was very broad and slurred, the conduction defect responsible for the lengthening of the QRS interval was probably located in the free wall of the right ventricle rather than in the septum. Activation of the anterior wall of the left ventricle was believed responsible for the small R wave in V_4 and V_5 and the small S wave in V_1 and V_2. Opposing negative potentials coming from activation of the right ventricle and perhaps the posterior wall of the left ventricle probably accounted for the smallness and brevity of the r wave in V_4 and V_5. The duration of the QS deflection in V_6 corresponded with the time interval from the end of the R to the termination of the QRS complex in V_4 and V_5. Thus, the QS deflection in V_6 was actually an S wave derived from activation of the right ventricle and perhaps in part from the posterior wall of the left ventricle. The potentials responsible for the antecedent qr deflection registered in V_4 and V_5 were apparently lost during the course of transmission through an emphysematous lung to position V_6.

Domelike elevation of the RS-T segment following a QS or a qrS complex may occur not only as a manifestation of myocardial infarction, but also from overdigitalization. The latter alternative was apparently responsible for the RS-T displacement in V_4, V_5 and V_6 and should have been suspected during life for the following reasons: (1) a full dose of Cedilanid had been given to a patient who had been receiving digitalis in unknown amounts; (2) the Q-T interval ranged from 0.23 second to 0.25 second and was slightly shorter than the Ashman and Hull standards of 0.25 to 0.28 second for the cycle lengths represented.[10] The absence of typical digitalis effects in other leads is left unexplained.

This case serves as another illustration of how closely patterns associated with right ventricular dilatation and hypertrophy mimic those due to myocardial infarction. An erroneous diagnosis of myocardial infarction was made by all members of the department who studied the tracing prior to knowledge of the pathologic findings. Experience with this case indicates that caution must be exercised in the interpretation of findings in leads from the left precordium when leads from the right precordium show evidence of right bundle branch block or right ventricular hypertrophy.

Summary

The differential diagnosis between uncomplicated bundle branch block and that associated with myocardial infarction has been brought out through a detailed analysis of the electrocardiograms in eleven cases. These cases were selected because of (1) QRS-T patterns considered diagnostic, or at least suggestive, of myocardial infarction; (2) subsequent exclusion of myocardial infarction at autopsy.

Left bundle branch block was present in four

of the cases and was manifested by one or more of the following signs that raised the question of myocardial infarction or led to an erroneous diagnosis during life: (1) broad, slurred QS deflections in the six customary precordial leads (V_1 through V_6); (2) a triphasic qrS pattern, consisting of small, narrow q and r deflections and a deep, broad S wave, in one or more precordial leads just to the right of the transitional zone; (3) registration of an rS complex in Lead V_1 and/or V_2 and a QS deflection in one or more leads farther to the left; (4) progressive diminution of the initial R wave, then replacement by a qRS deflection as the electrode was moved towards the left; (5) coveplane inversion of the T wave associated with QS- or W-shaped complexes in leads near the transitional zone.

Right bundle branch block was present in the other seven cases and was manifested by one or more of the following signs likely to be mistaken for those of myocardial infarction: (1) pseudo Q waves in leads from the right precordium and/or transitional zone; (2) elevation of the RS-T junction and abnormal inversion of the T wave in leads from the right precordium; (3) reduction in the R wave together with deep inversion of the T wave in intermediate leads; (4) concordant T waves; (5) prominent Q waves in leads from the left precordium.

Acknowledgment

The autopsy findings were furnished by Drs. B. E. Stofer and T. Hiratzka of the Department of Pathology. Dr. Howard Klein assisted in the collection of the electrocardiographic material. The tracings were retouched by Miss Evelyn Erickson and Miss Geraldine Chesney.

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Other QRS-T Patterns that May Be Mistaken for Myocardial Infarction

IV. Alterations in Blood Potassium; Myocardial Ischemia; Subepicardial Myocarditis; Distortion Associated with Arrhythmias

By Gordon B. Myers, M.D.

The electrocardiograms of patients in whom myocardial infarction was excluded at autopsy are presented to bring out (1) differentiation of patterns due to subendocardial ischemia or hypopotassemia from those due to subendocardial infarction; (2) differentiation of cove-plane T inversion associated with subepicardial myocarditis or ischemia from that accompanying myocardial infarction; (3) errors resulting from attempts to draw diagnostic inferences from the registration of prominent Q waves and/or cove negative T waves in ectopic beats or tachycardias.

THE DIFFERENTIATION between the precordial electrocardiogram of myocardial infarction and that associated with left ventricular hypertrophy, right ventricular hypertrophy and dilatation, and bundle branch block has been covered previously.[1–3] In this article, certain electrocardiographic features suggestive of myocardial infarction, but actually referable to alterations of blood potassium, to myocardial ischemia, to subepicardial myocarditis with or without pericarditis, or to QRS distortion associated with arrhythmias are brought out through the presentation of cases in which the possibility of myocardial infarction was excluded at autopsy. The method of study was similar to that described in previous reports.

Alterations in Blood Potassium

The electrocardiographic findings associated with both excessive elevation[4–8] and abnormal depression[9–11] of blood potassium have been described fully and need not be reiterated. The possibility of mistaking hypopotassemic depression of the RS-T junction and inversion of the T wave for acute subendocardial infarction will be brought out through the presentation of Case 39. The resemblance of certain features of the biphasic QRS-T complex associated with extreme hyperpotassemia to the pattern of extensive infarction with bundle branch block will be brought out through the presentation of Case 40.

Case 39.—The patient, a woman, age 45 years, was admitted to the hospital on June 18, 1943, in deep diabetic coma, accompanied by marked dehydration and profound peripheral circulatory collapse. The first electrocardiogram (fig. 1) obtained two hours after admission, when the carbon dioxide combining power was 10 volumes per cent, showed normal QRS complexes and tall, sharply peaked T waves. The latter finding was strongly suggestive of hyperpotassemia, but could have been due, in part, to the acidosis. During the first twenty-four hours, the patient received 1540 units of insulin, 1000 cc. of plasma, and 14,000 cc. of parenteral fluid, partly in the form of physiologic saline, partly as 5 per cent glucose. Peripheral circulatory collapse persisted until June 19 and normal blood pressures were maintained thereafter. No cardiac glycosides were given at any time to this patient.

The second tracing was obtained shortly after recovery from shock, when the carbon dioxide combining power was 50 volumes per cent and blood sugar 450 mg. per 100 cc. of blood. The QRS deflections showed slight reduction in voltage, but no significant change in configuration. On the other hand, striking changes occurred in the RS-T complexes, characterized by a 1-mm. depression of the RS-T segments and flattening of the T waves in Leads I and II; a reversal in the direction of T_3; a 2-mm. depression of the RS-T segments and inversion of the previously tall erect T waves in Leads V_4 and V_6; an apparent lengthening of the Q-T interval in Leads V_4 and V_6. These changes were attributed to acute ischemia or patchy infarc-

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tion of the subendocardial aspect of the anterolateral wall of the left ventricle, consequent upon the prolonged shock. The possibility of hypopotassemia was not considered at the time, but appears likely, in view of the recent reports of Holler[12] and of Martin and Wortman.[13] The severe dehydration antedating admission should have been accompanied by shift of potassium from cells to plasma, and should have resulted in elevation of blood potassium and marked loss in the urine. The excessive fluid intake during the first twenty-four hours should have caused further loss of potassium in the urine and the restoration of carbohydrate metabolism under the stimulus of large doses of insulin should have caused shift of potassium from plasma to liver, thereby reducing blood potassium to dangerously low levels.

The blood sugar reached normal levels on June 20, as a result of an additional 800 units of insulin during the second twenty-four-hour period and was maintained between 100 and 200 mg. per 100 cc. of blood thereafter by an average dose of 75 units of insulin daily. An additional 5000 cc. saline and glucose solution given on June 19 proved excessive, as shown by the appearance of edema and alkalosis. Repeat tracings on June 21 and 23 showed no significant change in QRS contour, but the voltage was reduced in Leads II, III, and V_4, probably owing to edema. The depressed RS-T segments progressively approached the isoelectric line and the inverted T waves became upright in Lead V_4 and isoelectric in V_6. The improvement in RS-T complex and T wave occurred despite the deterring influence of alkalosis,[14] as evidenced by a carbon dioxide combining power maintained at 80 volumes per cent, and was probably due to partial replenishment of potassium by oral feedings.

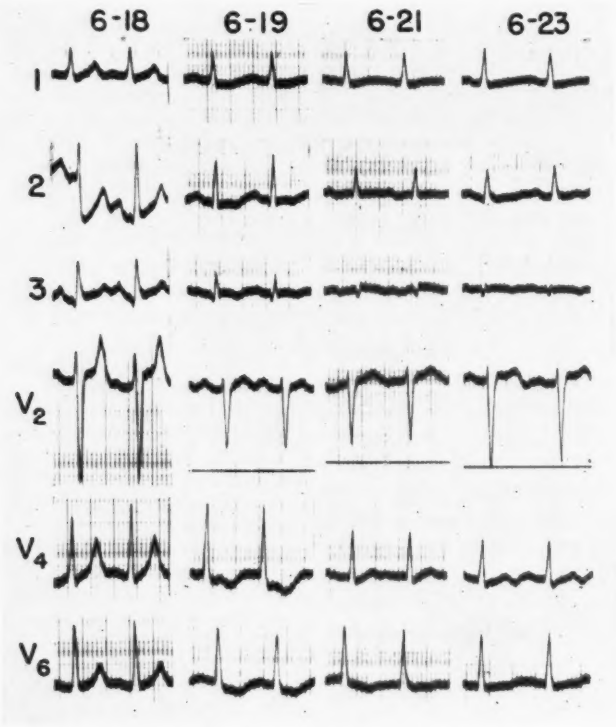

FIG. 1.—Serial electrocardiograms in Case 39.

Deep coma was replaced by stupor on June 19 and by drowsiness on June 20, but complete mental clearing failed to occur. After a four-day interval of drowsiness and irrationality, the patient lapsed into coma and died on June 25. The diabetes was under good control and moderate alkalosis was still present at the time of death. The principal autopsy finding was subcortical devastation necrosis, attributed to cerebral anoxia. The heart weighed 350 grams. The coronary vessels were normal by injection and dissection and no evidence of myo-

cardial infarction was found. The serial changes in the electrocardiogram were, in retrospect, probably the result of alterations of blood potassium.

Case 40.—The patient was a man, 37 years of age, who gave a history of diabetes mellitus of thirteen years' duration, intermittent dependent edema for five years, hypertension for two years, exertional dyspnea for one year, and blurring of vision for five months. Examination revealed hypertensive neuroretinopathy Grade 4, peripheral edema, albuminuria, and azotemia. The electrocardiogram of February 27, made shortly after admission and reproduced in figure 2, showed tall R waves and deeply inverted T waves in Leads V_5 and V_6 referable to left ventricular hypertrophy.

The hospital course was marked by a gradually increasing renal insufficiency up until the morning of April 16, when the patient awoke with generalized flaccid paralysis, profound circulatory collapse, and anuria. Electrocardiogram revealed auricular standstill, a QRS interval of 0.24 second, marked widening of the terminal portion of the QRS, and sharp peaking of the T wave typical of extreme hyperpotassemia. A distinct initial R wave was recorded in Leads V_1, V_2 and V_4, but a QS complex was present in V_3. This finding was considered analogous to the reduction or disappearance of the initial R wave sometimes found in leads at the transitional zone in uncomplicated bundle branch block[3] and hence did not constitute evidence of infarction.

The feature of hyperpotassemia that led to the inclusion of this case was the fusion of the terminal portion of the QRS with the T wave to make a pattern resembling that of infarction. This was best exemplified by Lead aV_L of the tracing of April 16. After the inscription of a small Q wave, a slightly prolonged upstroke and the first half of a precipitous intrinsicoid downstroke, the string veered at a right angle and then moved downwards in an arc, which at first glance resembled the elevated RS-T junction and covelike RS-T segment and T wave associated with recent myocardial infarction. However, from measurements of the QRS interval in other leads, it was evident that the RS-T junction was not at the right-angle bend, but rather at the notch in the lowermost point of the complex. Thus, the plateau following the right-angle bend and the subsequent descent were a part of the prolonged QRS and referable to the conduction defect of hyperpotassemia and were not a sign of infarction.

Death occurred seven hours after the last tracing. At autopsy the heart weighed 426 grams and showed slight left ventricular hypertrophy, but no evidence of infarction. The coronary vessels were of normal caliber and showed only minimal sclerosis. Renal sections showed far advanced intercapillary glomerulosclerosis of the type described by Kimmelstiel and Wilson.

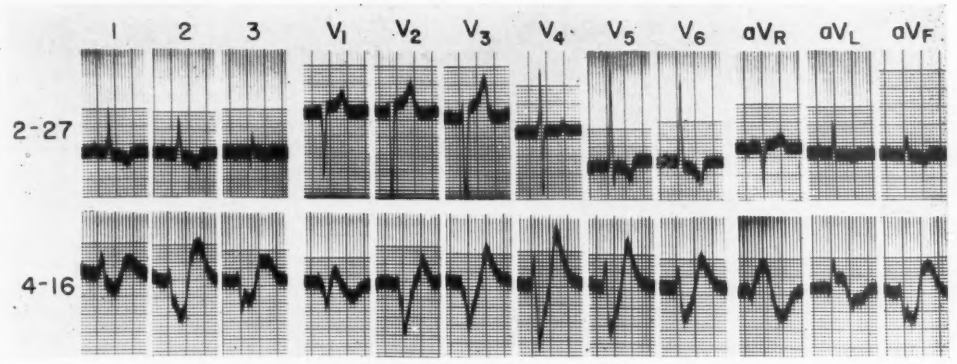

FIG. 2.—Serial electrocardiograms in Case 40.

ACUTE MYOCARDIAL ISCHEMIA

During acute myocardial ischemia, leads facing the epicardial surface of the involved area may show transitory depression of the RS-T segment and/or inversion of the T wave, usually without change in the QRS complex.[15–19] When the acute ischemia is confined to a portion of the circumference of the left ventricle, leads facing the opposite, uninvolved wall may display reciprocal elevation of the RS-T segment and/or tall erect T waves.[20,21]

Acute RS-T depression of similar magnitude and configuration may be observed in association with infarction or ischemia confined to the subendocardial layer of myocardium. Acute subendocardial ischemia may occur in previ-

ously normal hearts as a result of shock secondary to perforated peptic ulcer, hemorrhagic pancreatitis, intestinal obstruction, and other surgical conditions. When confronted with a

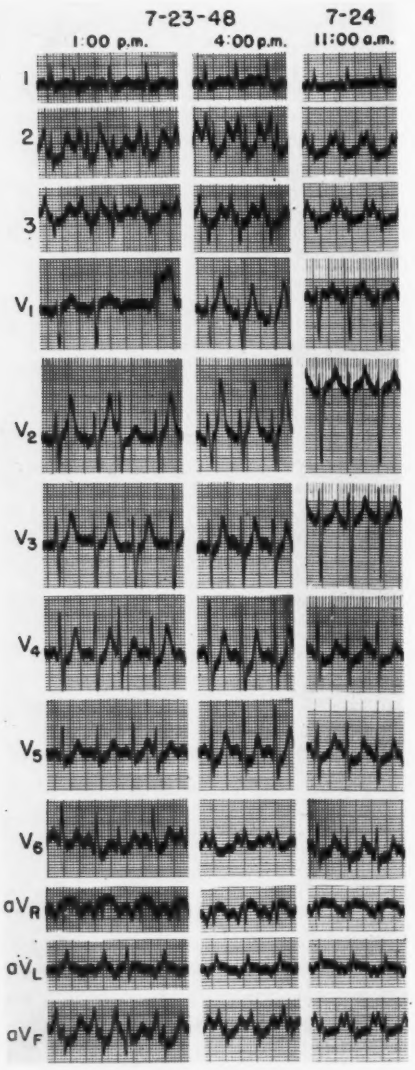

FIG. 3.—Serial electrocardiograms in Case 41.

clinical picture that raises the differential diagnosis between acute myocardial infarction and an acute surgical abdomen, the demonstration of abnormal RS-T depression and/or T-wave inversion, with or without reciprocal elevation of RS-T segments and exaggeration of T waves in leads from the opposite wall, does not establish the former nor exclude the latter alternative.[22–24]

The problem in differential diagnosis between acute subendocardial infarction and an acute surgical abdomen complicated by myocardial ischemia and the error that may result from overemphasis of electrocardiographic findings is exemplified by Case 41.

Case 41.—The patient was a 69 year old woman, who had had hypertension for several years, but was otherwise in good health until July 20, 1948, when she was awakened by severe epigastric pain, radiating retrosternally. The pain remained constant and no new symptoms appeared during the next three days. The patient was brought to the hospital on July 23 in shock with unobtainable blood pressure and cold, clammy extremities. The heart was not enlarged, and showed a mesodiastolic gallop and a rate of 130 with frequent premature beats. The abdomen was slightly distended, but revealed no tenderness, rigidity, or masses.

The first electrocardiogram (fig. 3) obtained on admission displayed sinus tachycardia with frequent auricular premature systoles, followed by aberrant ventricular response and rarely by dropped ventricular beat. The QRS complex of the sinus beats was not remarkable in contour in any lead, but that of the auricular premature beats in Leads aV_F, II and III was characterized by an equiphasic QR complex. These QR complexes conformed with the pattern of posterior infarction, as shown by a 0.03-second interval from onset to nadir of the Q wave in aV_F and a QR ratio of 100 per cent. This finding, together with the apparent 4-mm. RS-T depression in the cycles of sinus origin in Lead aV_F, led the resident to make a diagnosis of recent infarction of the subendocardial portion of the posterior wall, whereas the 1- to 2-mm. RS-T depression in Leads V_4, V_5 and V_6 was ascribed to continuation of the infarct into the subendocardial aspect of the anterolateral wall. The marked increase in RS-T depression in Leads V_4 and V_5 during the next three hours was interpreted by the resident as evidence confirming his impression of extension of the infarct into the subendocardial portion of the anterolateral wall, and the changes on the following day did not induce him to alter his diagnosis. The patient remained in shock and blood pressure was unobtainable except for brief periods after plasma transfusions. A terminal tracing at 1:00 P. M. on July 24 showed complete A-V block and death by ventricular standstill.

The tracings were studied by the author after death of the patient, but prior to knowledge of the autopsy, and a different interpretation was placed

upon the findings. Inasmuch as RS deflections were registered in all cycles of sinus origin in Leads aV_F, II and III, the abnormal QR deflections associated with ectopic auricular beats were not considered diagnostic of posterior infarction, since they could be explained by aberrant intraventricular conduction of the premature impulse, to be discussed later. The 4-mm. depression of the RS-T junction in aV_F was considered more apparent than real because of the tachycardia and the high voltage of the auricular complex. The heart was sufficiently rapid so that the beginning of the P wave was engrafted upon the end of the preceding T wave, thereby preventing return of the string to the isoelectric level. The extremely tall P wave in Lead aV_F was followed by an exceptionally deep T_a wave. The beginning of the T_a wave could be seen in front of the R wave and the remainder of the T_a wave could largely, if not entirely, account for the low level of the string following the S wave. Hence, the low level of the RS-T segment was regarded as a pseudodepression of no diagnostic significance.

The question arose as to whether the RS-T depression in the last three precordial leads could be explained entirely by tachycardia and the T_a deflection or whether it was due in part to a lesion in the subendocardial layer of the anterolateral wall. This question was settled by the marked increase in RS-T depression in V_4 and V_5 three hours later with little change in rate or P waves. The increasing RS-T depression in V_4 and V_5 was, therefore, indicative of a lesion of the subendocardial layer of the anterolateral wall of the left ventricle. The findings were attributed to acute ischemia, rather than to subendocardial infarction, because of an increase, rather than a decrease, in the amplitude of the initial R waves in Leads V_4 and V_5. The elevated RS-T junction and terminal inversion of the T wave in Lead aV_R were the result of cavity potentials reciprocal to those responsible for the findings in Lead V_6. The high-peaked T waves in Leads V_1 and V_2 aroused the suspicion of hyperpotassemia and the progressive narrowing of the base and sharpening of the apex of the T wave in the last two tracings, along with the lengthening of the Q-T interval, were best explained on this basis. Thus, the electrocardiographic findings were attributed in part to acute ischemia of the subendocardial portion of the anterolateral wall of the left ventricle and in part to hyperpotassemia. Unfortunately the blood potassium was not determined.

Autopsy revealed gangrene of the ileum as the cause of death and showed no evidence of myocardial infarction or coronary narrowing. The heart weighed only 200 grams. There was pallor of the subendocardial two-thirds of the left ventricular wall on gross examination and subendocardial interfibrillary edema without alteration of nuclei or striations was found on microscopic study of a number of blocks. These findings were attributed to myocardial ischemia and thus gave support to the revised interpretation of the electrocardiographic findings.

Cove-plane T waves, manifested by upward bowing of the RS-T segment, sharp inversion of the T wave, and a progressive evolution in serial tracings, are not pathognomonic of myocardial infarction, but may be found in association with acute myocardial ischemia,[25, 26] pericarditis,[27, 28] and other conditions.[1, 2, 3] The association of cove-plane T waves with acute myocardial ischemia is exemplified by Cases 42 and 43 and the association with pericarditis by Cases 44 and 45.

Case 42.—The patient was a janitor, 58 years of age, who had had intermittent epigastric pain, radiating into both sides of the chest, and increasing exertional dyspnea for a period of six months. There were physical signs of syphilitic aortic insufficiency, aneurysm of the ascending aorta, and hypersensitivity of the carotid sinus. No cardiac glycosides were given.

Electrocardiograms obtained on May 9, shortly after admission to the hospital, and repeated after a week of hospitalization, are reproduced in figure 4. The striking findings in the first tracing were the sharply inverted T waves and convex RS-T segments in the last five precordial leads, associated with isoelectric RS-T junctions and normal QRS complexes. The RS-T pattern in these leads was compatible with anterolateral infarction, myocardial ischemia, or pericarditis. The normal QRS complexes were strongly against the classical variety of anterolateral apical infarct, but were consistent with (1) the very rare infarct limited to the subepicardial layer of the apical portion of the anterolateral wall; (2) an ischemic zone beyond the border of an infarct localized to the basal portion of the anterolateral wall. High precordial leads should have been taken to investigate further this latter possibility.

The tracing of May 16 showed increase in R wave at the expense of the S in aV_L and in the last four precordial leads, probably due chiefly, if not entirely, to counterclockwise rotation. The QRS deflection was still considered within the limits of normal. A much greater change occurred in the RS-T segment and T wave in the last five precordial leads and could not be explained by the cardiac rotation. The previously inverted T waves had become low upright in the last five precordial leads, whereas the previously isoelectric RS-T junctions became considerably depressed in V_4 and slightly depressed in V_3 and V_5. The development of RS-T depression was strongly against pericarditis or subepicardial infarction, but consistent with ischemia of the apical portion of the anterior wall. High precordial leads

should also have been taken on this date to rule out the possibility that the apical ischemic zone was associated with an infarct localized in the base of the anterolateral wall.

Death occurred three weeks later. At autopsy the heart weighed only 393 grams. Syphilitic aortic insufficiency, aneurysm of the ascending aorta, and narrowing of the coronary ostia were found. The epicardium was normal and no evidence of infarction was found on gross examination or in the twelve microscopic blocks studied. However, a patchy fibrosis was found in the majority of these blocks and was believed secondary to coronary insufficiency. Thus, autopsy excluded the possibility of myocardial infarction and pericarditis, but revealed changes which suggested that myocardial ischemia was responsible for the electrocardiographic abnormalities.[29]

Case 43.—The patient was a 15 year old school girl, who gave a history of recurrent episodes of anemia since the age of six. She was admitted to the hospital in a severe relapse. The anemia was established as hemolytic in type by fecal stercobilin excretion ranging from 720 to 1042 mg. in twenty-four hours. Sarcoidosis was discovered on bone marrow examination and was confirmed by lymph glandular biopsy. No cardiac glycosides were given.

The electrocardiogram of Oct. 31, 1944 (fig. 5), was obtained during the severe relapse, when the red blood count was 1,050,000 per cu. mm. of blood and the hemoglobin was 20 per cent. The QRS complexes were normal in all leads. The RS-T junctions were isoelectric throughout. The T waves were flattened in Leads V_6 and I. The striking feature was the sharp inversion of the T waves in Leads V_2 and V_4. Although the RS-T segments and T waves in these leads mimicked the cove-plane pattern of myocardial infarction, no consideration was given to such a diagnosis in the clinical interpretation because of the normal QRS complexes and the age of the patient. The possibility that the inverted T waves represented a normal variant of adolescence was deemed unlikely because of the depth of the T waves in a lead as far to the left as position V_4 and was subsequently excluded by the disappearance of the inversion.

With the aid of transfusions, a progressive rise in red blood and hemoglobin occurred during November, to reach levels of 3,650,000 per cu. mm. and 62 per cent, respectively, on December 2. Parallel changes in T waves occurred, characterized by progressive increase in amplitude in Leads V_6, I and II, by metamorphosis of the deeply inverted T wave in V_4 to a normal upright deflection, and by change from a deeply inverted to diphasic to flattened T wave in V_2. No significant alteration occurred in the normal QRS complexes. Because of the parallelism between improvement in T waves and blood count, it was concluded that the initial cove-plane inversion was most likely an expression of acute myocardial ischemia secondary to the hemolytic crisis.[30] The possibility of acute cor pulmonale was given serious consideration in the differential diagnosis, but was deemed less likely because of the deeper inversion of the T waves in Lead

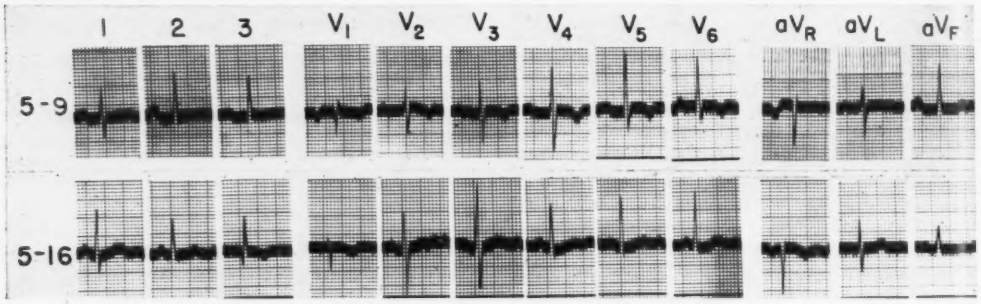

FIG. 4.—Serial electrocardiograms in Case 42.

V_4 than in V_2. An intercurrent pericarditis was a further alternative that could not be excluded by electrocardiographic findings.

Splenectomy was performed on December 5, but death occurred four weeks later in another hemolytic crisis. The diagnosis of sarcoidosis was confirmed at autopsy. The heart and coronary arteries were entirely normal to gross and microscopic examination. The negative pathologic findings constituted indirect evidence supporting the antemortem ascription of the transiently inverted T waves to acute myocardial ischemia secondary to acute hemolytic anemia.

PERICARDITIS AND SUBEPICARDIAL MYOCARDITIS

Although the RS-T pattern associated with pericarditis was described in 1929[31, 32] and repeatedly elaborated upon during the next decade,[33–37] reports continue to appear of cases with

electrocardiograms typical of pericarditis, in which the authors made a diagnosis of "coronary occlusion" or myocardial infarction and failed to mention the possibility of pericarditis.[38–40] This indicates the need for a brief resume of the electrocardiographic findings in pericarditis and re-emphasis of the features that

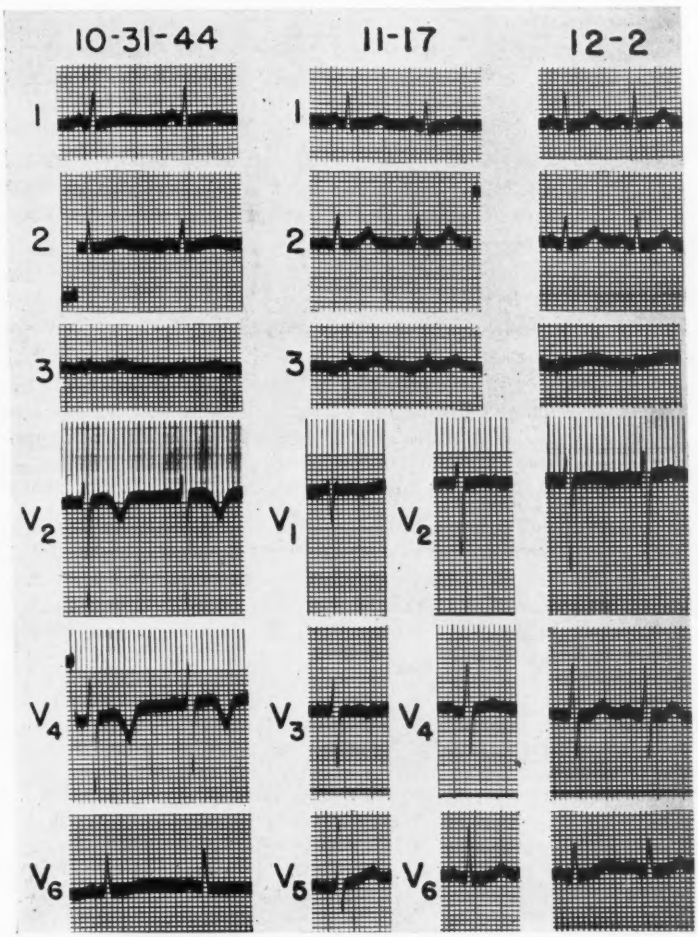

FIG. 5.—Serial electrocardiograms in Case 43.

may be mistaken for those of myocardial infarction.

The RS-T changes about to be described are an expression of a lesion of the superficial layers of myocardium,[33–37,41] rather than an involvement of the epicardium per se, but, through custom, are designated as the pattern of pericarditis. During the acute stage, the RS-T junction is characteristically elevated and is followed by a monophasic upright T wave. At first, the RS-T segment usually ascends in a straight line or in a curved line with upward concavity, but may exhibit an upward convexity similar to that in myocardial infarction. As the acute passes into the subacute stage, serial tracings reveal a progressive return of the RS-T junction to the isoelectric line, accompanied by a change to an upwardly convex, domelike RS-T segment, and a deepening coveplane inversion of the T wave, similar to that in myocardial infarction. As the lesion heals,

there is a progressive return to normal upright T waves.

The RS-T and T pattern of pericarditis, both in individual and in serial tracings, may be identical with that accompanying acute myocardial infarction. Certain minor differences in the distribution of the abnormal T waves and in their rate of evolution may aid in the electrocardiographic differentiation of pericarditis and myocardial infarction. In the former, comparable RS-T patterns are usually found in leads facing the anterior and posterior ventricular surface, owing to the tendency towards generalization of pericarditis; in the latter, reciprocal RS-T patterns are characteristically present owing to the tendency for localization of infarction to one wall. The evolution of the RS-T changes is usually much more rapid in pericarditis than in myocardial infarction.

The most important criterion in differential diagnosis is the direction of the initial deflection of the QRS wave. An abnormal Q wave is usually demonstrable in an overlying lead within a few hours after infarction of the subjacent wall, persists throughout organization of the lesion, and often remains permanently. On the other hand, Q waves are not produced by pericarditis or subepicardial myocarditis; however, a pre-existent Q wave may persist after the development of pericarditis and may give rise to diagnostic difficulties. In pericarditis, the R wave may be reduced in voltage, but remains normal in contour; in myocardial infarction, abnormal reduction in the R wave is characteristically present in leads facing the lesion and is often accompanied by abnormal slurring, notching or widening of the QRS complex.

In spite of the foregoing criteria, pericarditis and myocardial infarction may be confused electrocardiographically. Diagnostic difficulties may be encountered in the presence of the following three patterns: (1) Localized abnormal elevation of the RS-T junction and/or cove-plane inversion of the T wave in the presence of an initial R wave of normal or low voltage. Such a pattern is characteristic of a lesion of the subepicardial layer of myocardium and is usually not infarctive in origin. Before reaching a diagnosis of pericarditis, however, it is necessary to consider the rare infarct limited to the subepicardial layer and the less rare situation in which there is a widespread pericardial reaction secondary to a relatively small infarct localized to a portion of the wall not subtended by the customary precordial and limb leads. To further investigate the latter, supplementary leads should be obtained from the V_7 and V_8 positions and from the upper precordium and left axilla at the horizontal level of the junction of third intercostal space and sternum. (2) Abnormal elevation of the RS-T junction and/or cove-plane inversion of the T wave, in complexes exhibiting a distinct Q wave and a normal to borderline Q/R ratio. This combination brings up the differentiation between pericarditis in a person with pre-existent Q waves and infarction involving a thin layer of subendocardial muscle and extending through the wall in patchy fashion to reach the subepicardial layer. (3) Localized reduction in the amplitude of the R wave in a given lead below that attained in adjacent leads to the right and left. Such a finding suggests of patchy infarction, but may be associated with pericardial thickening or effusion. Four cases of pericarditis (Cases 44 to 47, inclusive), that collectively exemplify the foregoing three patterns, will be presented to bring out the features that may be mistaken for those of myocardial infarction.

Case 44.—The patient was a girl 19 years of age. She was admitted to the hospital on May 15 with a history of transitory sore throat three weeks previously and migratory joint pain and swelling of one week's duration. A pericardial friction rub was audible during the first week in the hospital, but no evidence of pericardial effusion was detected at any time. Embolic phenomena were first observed on June 3, signs of aortic insufficiency were detected on June 8, and congestive failure developed on June 13. No cardiac glycosides were given until June 14, when 0.8 mg. Cedilanid was injected intravenously one hour prior to the tracing.

Serial electrocardiograms are reproduced in fig. 6*A*. The tracing of May 15 showed elevated dome-like RS-T segments and sharply inverted T waves in the last four precordial leads and in all three limb leads, simulating the cove-plane contour of recent myocardial infarction. However, the normal initial R waves in each of these leads indicated that the lesion was probably confined to the sub-

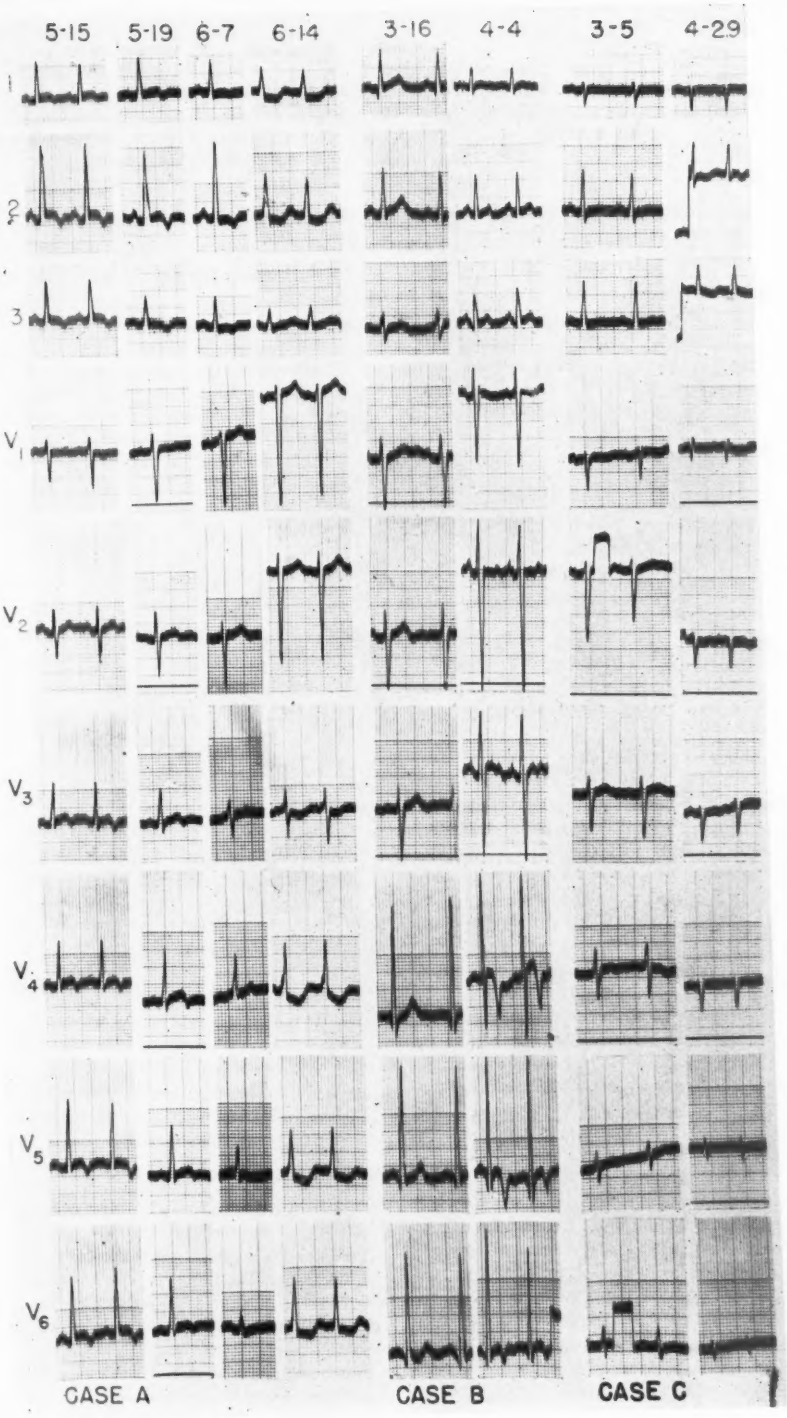

FIG. 6.—Serial electrocardiograms in pericarditis and subepicardial myocarditis. *A*, Case 44; *B*, Case 45; *C*, Case 46.

epicardial layer and thus were strongly in favor of pericarditis and against myocardial infarction. To exclude more definitely the possibility of localized high anterior, high lateral or posterior infarction, additional leads are usually advisable from the back, upper precordium and axilla. Such a procedure was unnecessary in this case, in view of the clinical picture.

The last four precordial leads on May 19 showed pronounced domelike RS-T elevation and a sharp, though shallow, inversion of the terminal portion of the T waves. This RS-T pattern simulated that associated with recent myocardial infarction, but could also be ascribed to a superficial lesion because of the normal initial R waves. The regression in RS-T abnormalities and reduction in the voltage of the R waves in the tracing of June 7 were consistent with the evolution in pericarditis. On the other hand, the marked RS-T depression and the slurring and prolongation of the ascending limb of the R waves, which appeared in Leads V_4, V_5, V_6, I and II on June 14, were not attributable to pericarditis. The contour of the RS-T segments was suggestive of digitalis action, but the degree of depression was much greater than is customarily observed after doses of 0.8 mg. Cedilanid. Furthermore, the fact that the Q-T interval was beyond the upper limits of normal and relatively longer than in previous tracings indicated that Cedilanid was not the major factor in the RS-T changes. From the combination of slurring and prolongation of the ascending limb of the R waves with RS-T depression and Q-T lengthening, it was postulated that the subepicardial myocarditis had extended through in patchy fashion to the subendocardial layer.

Death occurred three hours after the last tracing. At autopsy the heart weighed 390 grams and showed evidence of a diffuse fibrinous pericarditis, without effusion, and an acute vegetative aortic valvulitis, superimposed on acute rheumatic endocarditis. The epicardial inflammatory reaction had extended into the superficial layer of myocardium. In addition, there was an acute rheumatic myocarditis distributed in patchy fashion through the deeper layers of myocardium. This was believed to have been the major factor in the terminal changes in the R waves and RS-T complexes in left precordial leads.

Case 45.—The patient was a laborer, 30 years of age, who gave a history of rheumatic fever complicated by a valvular lesion in 1939. He was admitted to the hospital on March 16, 1946, because of sudden hemiplegia. Examination revealed left ventricular hypertrophy, aortic insufficiency, embolic phenomena and positive blood cultures for *Streptococcus viridans*.

The tracing taken shortly after admission on March 16 is reproduced in figure 6*B* along with a repeat electrocardiogram obtained on April 4. The last three precordial leads on March 16 displayed Q waves averaging 3 mm. in depth, R waves ranging from 28 to 32 mm. in height, upwardly concave RS-T segments, and normal erect T waves. Since the time interval from onset to nadir of the Q waves did not exceed 0.02 second and since the Q/R ratio was in the neighborhood of 10 per cent, it was concluded that the Q waves were normal. The possibility of left ventricular hypertrophy was suggested by the high voltage of the R waves, but a diagnosis of such could not be made electrocardiographically, in view of the normal duration of the ascending limb of the R wave and normal contour of RS-T segments and T waves.

The patient failed to respond to large doses of penicillin. Acute pulmonary congestion develped on April 2, necessitating rapid digitalization. Unfortunately, a repeat electrocardiogram was not obtained until April 4. Leads V_4 and V_5 showed very striking changes from upwardly concave to convex, domelike RS-T segments, and from normal erect T waves to deeply and precipitously inverted, narrow-based T waves. In Lead V_6, a less deep, cove-plane inversion took place. The possibility of overdigitalization was considered because of the short Q-T interval and the demonstration of cove-plane inversion of the T waves after toxic doses of digitalis in animals,[42] but was discarded because of the normal P-R interval and the lack of typical digitalis effects in other leads. The T wave pattern in V_4 and V_5 was the reciprocal of that associated with hyperpotassemia, but the lack of typical changes in other leads excluded such a possibility. The fact that the deep T-wave inversion was confined to Leads V_4, V_5 and V_6 pointed to a localized lesion in the anterolateral aspect of the left ventricle. The presence of Q waves 3 mm. deep in V_6 and 3 to 5 mm. deep in V_5 might have led to a diagnosis of recent anterolateral infarction, were it not for the extremely tall succeeding R waves on April 4 and the presence of a comparable QR pattern in the previous electrocardiogram. The fact that the time interval from onset to nadir did not exceed 0.02 second, coupled with the QR ratios below 15 per cent in the tracing of April 4, indicated that the Q waves were comparable to those in the previous tracing and were not attributable to infarction. As a consequence, a diagnosis of localized pericarditis was made.

Death occurred on April 7. Autopsy revealed subacute vegetative aortic valvulitis and widespread miliary abscesses of embolic origin. The heart weighed 521 grams and showed evidence of left ventricular hypertrophy. The coronary arteries were normal. There was no evidence of myocardial infarction and no gross signs of pericarditis. However, microscopic examination revealed an acute subepicardial myocarditis, which was believed responsible for the RS-T changes.

Case 46.—The patient, a Negro man, 23 years of age, was hospitalized from February 22 to May

14 with a plateau fever, averaging 101 F. Six-foot roentgenograms showed a 59 per cent cardiothoracic ratio on admission, no change on March 20, and an increase to 75 per cent on April 26. On the latter date, there were typical physical signs of pericardial effusion, which proved to be serosanguinous in type. Miliary infiltration was first detected in the lung fields on April 26; the lesions increased in size in subsequent roentgenograms.

The first tracing obtained on March 5 (fig. 6*C*) showed an initial upstroke in all precordial leads. This R wave increased progressively from an amplitude of 1 mm. in V_1 to 6 mm. in V_4, then fell off to 4 mm. in V_5 and increased to 6 mm. in V_6. In view of the equiphasic RS complex in V_5, the localized reduction in the amplitude of the R wave was but measured 1 to 2 mm. in amplitude in Leads V_3 and V_5. The localized reduction in the amplitude of the R wave in V_4 could not be dismissed as a transitional zonal phenomenon because of the deep S wave, which exceeded the overall voltage of the equiphasic RS deflection in V_5. The absolute and relative amplitudes of the R waves, as observed in the precordial leads of this case, would ordinarily raise the question of a healed patchy anteroseptal infarct. This error was avoided in this instance because the clinical findings were known at the time of electrocardiographic interpretation.

The patient died of miliary tuberculosis on May 14. At autopsy the heart weighed 493 grams and showed a universal plastic tuberculous epicarditis, which reached a maximal thickness of 1 centimeter.

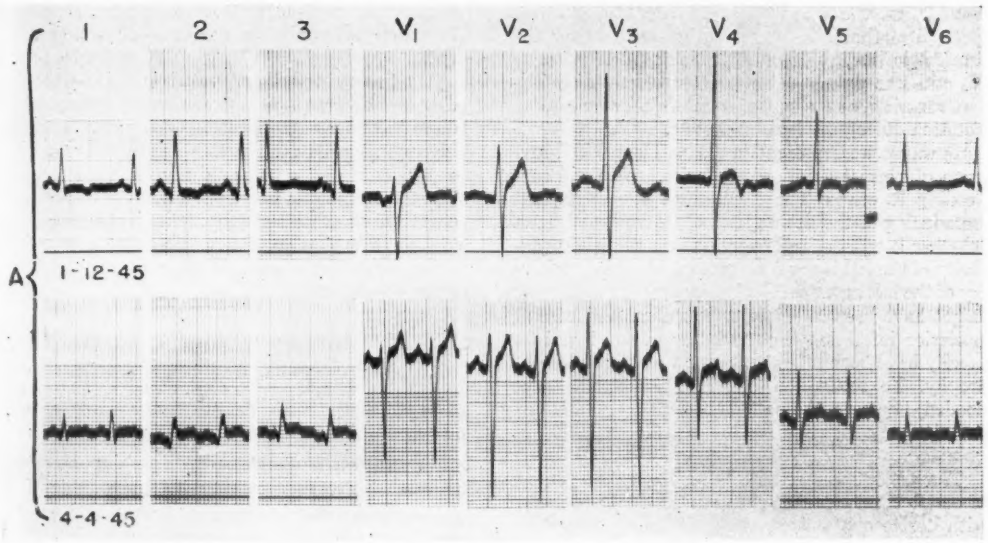

FIG. 7.—Serial electrocardiograms in Case 47.

considered a transitional zonal phenomenon. The T wave was isoelectric or low upright in all leads except V_4, where there were respiratory fluctuations between a low upright and shallowly inverted deflection. The significance of the T-wave patterns was not appreciated at the time, but in retrospect it seems likely that they were referable to pericarditis.

The tracing of April 29 showed a generalized reduction in QRS voltage, shallow inversion of T waves in Leads II and III, and flattening of the T waves in all other leads, consistent with pericardial effusion or constrictive pericarditis. The noteworthy feature of the tracing, which was responsible for the inclusion of the case in this article, was the localized reduction in the amplitude of the R wave in Lead V_5. The R wave was barely detectable in this lead, The pericardial sac contained 250 cc. of serosanguinous fluid. The generalized and localized reduction in R wave could be correlated with the pericarditis.

Case 47.—The patient was a man 57 years of age, who had had hypertension for three years. He was first admitted to the hospital on Jan. 11, 1945, because of sudden vertigo. No cardiac glycosides were given. Electrocardiogram made on the following day and reproduced in fig. 7 showed initial R waves in all precordial leads. Those in Leads V_1 and V_2 were followed by deep, slightly widened S waves, elevated RS-T junctions, and tall upright T waves consistent with the changes produced by left ventricular hypertrophy in right precordial leads. The R waves in left ventricular Leads V_5 and V_6 were not abnormal in amplitude or duration, but

were followed by isoelectric RS-T junctions and inverted T waves suggestive, but not diagnostic, of left ventricular hypertrophy.

Lead V_4 required special attention because of domelike elevation of the RS-T segment and cove-plane inversion of the T wave. The tall initial R wave was against myocardial infarction and the limitation of the cove-plane pattern to one lead was against pericarditis. The fact that the R wave in V_4 resembled that in V_5 and V_6, whereas the S wave was comparable to that in V_3 and V_2, suggested that the electrode at position V_4 was in the vicinity of the transitional zone and that the RS-T complex represented an approximately equal admixture of effects from the two ventricles. The first portion of the elevated RS-T segment resembled that in Leads V_3 and V_2 and was probably transmitted chiefly from the right ventricle, whereas the terminal portion of the T wave, which extended below the isoelectric line, resembled the inverted T wave in V_5 and was probably transmitted chiefly from the left ventricle, as a result of counterclockwise cardiac rotation during mechanical systole.

Q waves were present in Leads II and III, and were of normal amplitude in reference to the succeeding R waves. The significance of slurring and broadening of the downstroke of Q_3 depended upon whether it was derived from prolonged initial negativity of the left leg or from prolonged initial positivity of the left arm.[43] In the former event, the slurring might have been referable to healed subendocardial posterior infarction; in the latter event, it might merely have been referable to left ventricular hypertrophy. To settle the question, Lead aV_F was needed; however, the patient became symptom-free and left the hospital before this lead could be obtained.

The patient was symptom-free until the evening of April 3, 1945, when he was suddenly stricken with severe oppressive pain, confined to the retrosternal region, complicated by shock with blood pressure of 90/80. The electrocardiogram of April 4 was obtained fourteen hours after the onset of the pain. In the preliminary study, made without access to the previous tracing, a tentative diagnosis of recent posterior infarction was reached from the following findings in Leads aV_F, II, and III: a QR ratio of 25 to 40 per cent, elevation of the RS-T junctions and monophasic upright T waves. Complicating anterolateral pericarditis was postulated from the elevation of the RS-T segments and notched T waves in V_4, V_5, and V_6 in the presence of normal initial R waves.

Upon re-examination of the second tracing in the light of the original, further support to the diagnosis of recent anterolateral pericarditis was obtained from the decrease in amplitude of the R waves without change in contour of the QRS complex in the last three precordial leads. On the other hand, comparison of the two sets of limb leads altered the interpretation of the second tracing. The Q waves were a remnant of those present in the first tracing and had shown a decrease in amplitude, rather than the increase expected from intercurrent posterior infarction. The downstroke of the Q wave in Lead aV_F was neither slurred nor prolonged. The reduction in the R waves was comparable to that in V_6 and, along with the RS-T elevation, could be explained by pericarditis, in the absence of infarction. Although the error made in the original interpretation of the second tracing was corrected by restudy in the light of a previous tracing, this case serves to exemplify the difficulties in differentiation of the patterns of acute pericarditis and myocardial infarction. This history of sudden severe retrosternal pain followed by shock, together with a QRS-T pattern indicative of pericarditis rather than myocardial infarction, pointed towards hemopericardium from ruptured dissecting aneurysm.

Death occurred twenty-six hours after the last electrocardiogram. Autopsy revealed hemopericardium secondary to dissecting aneurysm. There was marked concentric left ventricular hypertrophy, but no evidence of recent or old myocardial infarction. The coronary vessels showed no signs of compression or occlusion. The autopsy findings adequately explained the history of sudden severe retrosternal pain followed by shock and the electrocardiographic signs of pericarditis.

Distortion of the QRS Complex Secondary to Arrhythmias

QRS complexes resembling the pattern of myocardial infarction may be found in certain arrhythmias, as a result of (1) an ectopic ventricular focus, (2) aberrant intraventricular conduction of a premature impulse of supraventricular origin, (3) fusion of an inverted auricular deflection with the beginning of the ventricular complex. The remaining cases have been selected to exemplify the foregoing situations.

Ectopic Ventricular Beats.

Attention to the possible diagnostic significance of the QRS-T pattern in ectopic ventricular beats has been directed by Dressler's case of posterior infarction.[44] The QRS-T pattern in beats of sinus origin was typical of left bundle branch block and was not indicative of infarction. The diagnosis was made from the presence of a deep Q wave, elevated RS-T junction and cove-negative T wave in ectopic beats in Lead III and was subsequently con-

firmed at necropsy. Since then, another case of left bundle branch block has been reported,[45] in which the diagnosis of anterior myocardial infarction was made from the QRS-T pattern of ectopic ventricular beats in precordial leads. On the other hand, an impulse originating from an ectopic ventricular focus directly beneath the precordial electrode may give rise to a QS or QR complex, an elevated RS-T segment, and inverted T wave, irrespective of the presence or absence of myocardial infarction. The error that might result from an attempt to draw diagnostic inferences from the QRS-T pattern in ventricular ectopic beats is illustrated by Case 48.

Case 48.—The patient, a woman 38 years of age, gave a history of fever and night sweats of three months duration and had physical and roentgen signs of pericardial effusion. Three hundred and fifty cc. of straw-colored fluid were removed prior to the electrocardiogram reproduced in figure 8.

A sinus mechanism was present, complicated by intermittent premature ventricular beats with short episodes of bigeminy. The QRS complexes of sinoauricular origin were consistent with a diagnosis of pericarditis, because of (1) initial R waves that were low in voltage, but normal in configuration in all leads; (2) T waves that were inverted in V_2, V_3, V_4, and V_5 and isoelectric in the remaining leads. On the other hand, the QRS complexes of the premature ventricular beats in Lead V_3 (second, fourth, and sixth cycles) were characterized by a deep, wide, slurred Q wave, a domelike RS-T segment and inverted T wave, and were comparable to those in Dressler's case.[41] Although the presence of such a pattern in beats of sinoauricular origin would have been considered pathognomonic of infarction, its limitation to ectopic ventricular beats nullified its diagnostic significance in the antemortem interpretation of the tracing.

Death occurred three weeks after the electrocardiogram. Autopsy revealed tuberculous pericarditis. The epicardium was approximately 1 cm. in thickness. The inflammatory process extended into the subepicardial zone, but spared the deeper layers of myocardium. There was no gross or microscopic evidence of myocardial infarction. The deep, broad Q wave in the premature ventricular beats of Lead V_3 was probably a manifestation of an ectopic focus in the subepicardial layer.

Ventricular Tachycardia.

In the interpretation of electrocardiograms exhibiting sinus rhythm complicated by premature ventricluar beats, one is less likely to err by disregarding the configuration of ectopic beats and depending entirely upon the QRS-T pattern of intervening sinus beats. When sustained ventricular tachycardia is present, there are no sinus impulses to serve as a guide and

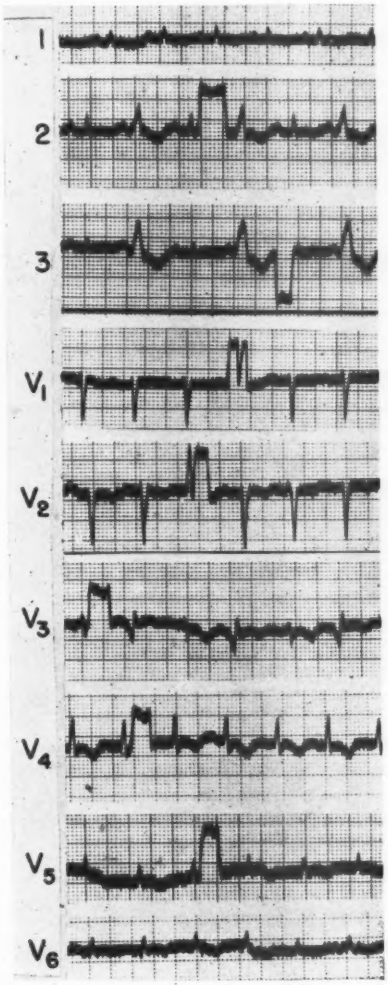

FIG. 8.—Electrocardiographic findings in Case 48.

one may be tempted to draw diagnostic inferences from QS or QR patterns, particularly in view of the fact that myocardial infarction is the commonest cause of this type of arrhythmia. The error from such deductions is exemplified by Case 49.

Case 49.—The patient was a man, 53 years of age, who began to have exertional and paroxysmal nocturnal dyspnea in the summer of 1942 and dependent edema the following autumn. He was admitted to the hospital with severe congestive failure in Jan. 1943. There was electrocardiographic evidence of left ventricular hypertrophy and nodal tachycardia, which later reverted to sinus rhythm. The patient was well compensated at discharge and was not again under observation until Sept. 18, 1943, when he was brought to the hospital in coma. No history was obtainable. Cedilanid 1.6 mg. was given intravenously prior to the electrocardiogram.

The tracing of September 18 revealed no evidence of auricular activity, a regular ventricular rhythm of 150 per minute, and a QRS interval of 0.14 second (fig. 9). Two explanations were considered: (1) auricular fibrillation complicated by ventricular tachycardia, (2) middle nodal tachycardia with defective intraventricular conduction. When the tracing was first examined, the latter alternative was favored partly because of the known presence of nodal tachycardia on the previous admission, but chiefly because of the absence of distinct f waves. Proceeding upon the assumption of a middle nodal tachycardia, the deep broad Q waves, late R wave, and RS-T elevation in Leads V_1 and V_2 were construed as evidence of septal infarction with right bundle branch block and the W-shaped QRS complexes in V_3 and V_4 were attributed to continuation of the lesion into the anteroseptal wall of the left ventricle. The QRS alternans in V_2, V_3 and V_4 was also considered a manifestation of anteroseptal infarction.

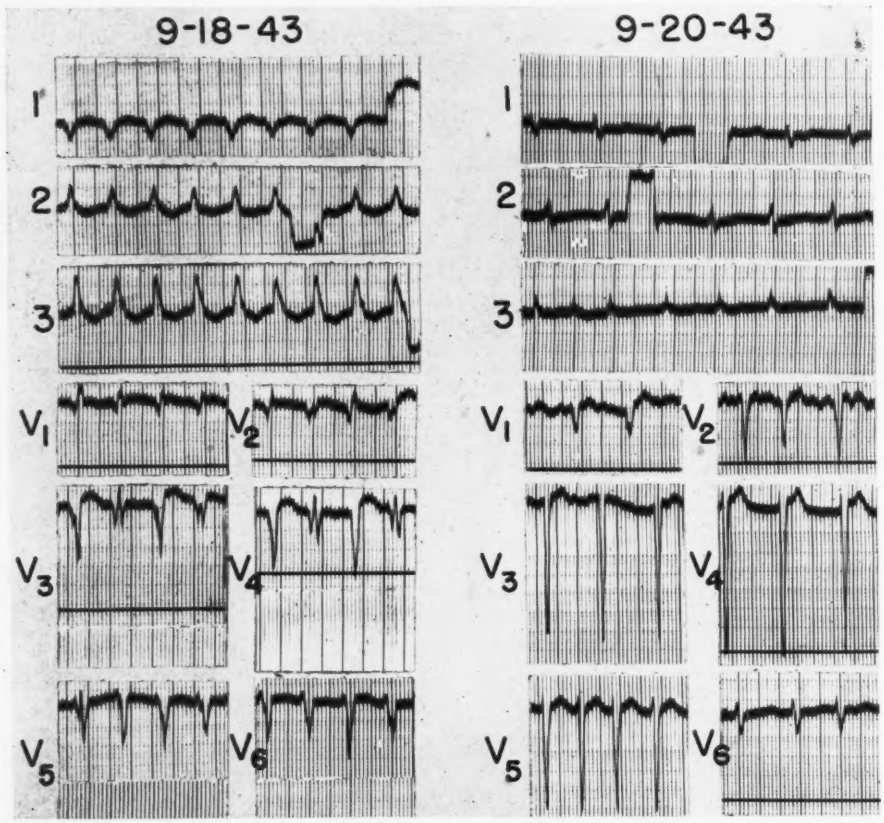

FIG. 9.—Electrocardiographic findings in Case 49.

The findings on September 20 (fig. 9) placed a different light both on the arrhythmia and the ventricular lesion. Distinct f waves were visible in V_1 and V_2 and the ventricular rhythm was totally irregular, indicating the presence of auricular fibrillation. The QRS interval had shortened to 0.10 second. All six precordial leads displayed an rS deflection. In the first three leads, the initial r

was minute and was followed by a broad, slurred S wave; in the last three leads, the r wave increased progressively at the expense of the S wave. The QS deflection in the first five leads indicated predominant transmission of the potential variations of the right ventricle to the left precordium; the small, amost equiphasic RS complex in V_6 represented a manifestation of the transitional zone. The wide transmission of the potential variations of the right ventricle over the left precordium was due in part to right ventricular hypertrophy, in part to clockwise rotation of the heart. The depth and breadth of the S wave in these leads indicated left ventricular hypertrophy. Leads V_7 and V_8 were not obtained in this case, but would probably have furnished confirmatory evidence of left ventricular hypertrophy in the form of a prominent R wave with late peak, reciprocal to the S wave of Lead V_2.

In view of the findings on September 20, reconsideration was given to the tracing of September 18 and the diagnosis was changed to auricular fibrillation complicated by A–V block and ventricular tachycardia. Since the deep broad Q waves in the first four precordial leads on September 18 gave way to a distant initial R wave on September 20, when the ventricles were activated by impulses of auricular origin, it was concluded that these Q waves were referable to an ectopic ventricular focus rather than to myocardial infarction. Both the ventricular tachycardia and QRS alternans probably represented manifestations of digitalis intoxication and could have been produced by the intravenous injection of 1.6 mg. Cedilanid, particularly if the patient had been receiving cardiac glycosides prior to admission.

Death occurred on September 21. At autopsy the heart weighed 690 grams and exhibited left ventricular hypertrophy and secondary right ventricular dilatation and hypertrophy. The coronary arteries were of normal caliber and no evidence of myocardial infarction was found. Thus, an erroneous diagnosis consequent upon the present of ventricular tachycardia was averted by a second tracing, taken after resumption of ventricular activation by impulses of auricular origin.

Aberrant Intraventricular Conduction of Premature Auricular Beats

This has been exemplified by the first tracing obtained on Case 41 (fig. 3). Premature auricular beats in Leads aV_F, II and III were followed by QR deflections that conformed with the pattern of posterior infarction both in respect to the duration of the descending limb of the Q wave and the relative amplitudes of the Q and R waves. A diagnosis of posterior infarction would have been justified if beats of sinoauricular origin in the same leads had displayed comparable QR deflections. Since RS deflections were registered in all cycles of sinus origin in Leads aV_F, II and III, the question arose as to whether the QR complexes associated with the ectopic auricular beats were manifestations of aberrant conduction in the absence of myocardial infarction or other ventricular lesion. Since repolarization is normally completed earlier in the anterior than in the posterior wall of the ventricles, it is possible for a premature auricular impulse to reach the ventricles immediately after the anterior wall has passed out of the refractory state, but while the posterior wall is still unresponsive. During the period while the impulse is activating the anterior, but is held up in the posterior wall, negative cavity potentials are transmitted through the posterior wall to the surface, to be recorded as abnormal Q waves in Leads aV_F, II and III. The fact that the ectopic auricular beats in Patient 41 were so premature that the ventricular response began 0.05 second prior to the end of the T wave of the preceding cycle indicated that aberrant intraventricular conduction in the absence of infarction could have accounted for the abnormal Q waves in Leads aV_F, II and III of the ectopic beats. Autopsy excluded the possibility of infarction, but did reveal an ischemia of the entire circumference of the subendocardial layer of the left ventricle. Whether or not the ischemia accentuated the normal difference in time of recovery from the refractory state in the anterior and posterior walls is unknown.

Fusion of a P Wave with the First Portion of the QRS Complex

The possibility of mistaking an inverted auricular deflection for a Q wave has been mentioned,[46] and is exemplified by Case 50.

Case 50.—The patient, a clerk 49 years of age, was admitted to the hospital on April 9, after three weeks of incapacitation because of progressive orthopnea, complicated by repeated hemoptyses. Past history disclosed exertional dyspnea for five years and retrosternal burning on walking against a cold wind during the preceding winter. There were physical signs of mitral and aortic stenosis and insufficiency.

The electrocardiogram reproduced in figure 10 was one of a series and was obtained after digitalization. Since the interpretation of this tracing would have been rendered more difficult if Lead V_{3R} had not been available, an analysis will first be made of the remaining leads. Casual inspection of Lead amplitude. In the remaining precordial leads, the QRS began with an upstroke and the R wave steadily increased at the expense of the S wave as the electrode was moved towards the left. The R/S ratio thus failed to reveal evidence of preponderant hypertrophy of the right ventricle.

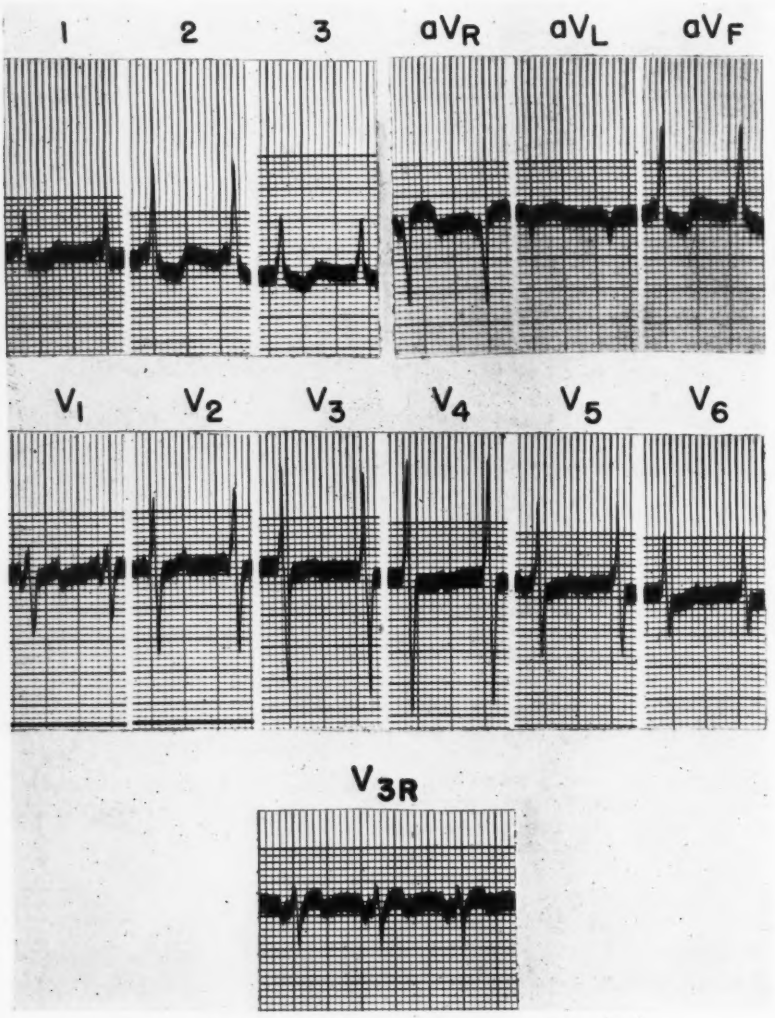

FIG. 10.—Electrocardiographic findings in Case 50.

V_1 showed a precipitous initial downstroke, 2 mm. in depth and 0.02 second in duration, followed by a splintered R wave reaching 3 mm. above the isoelectric line and then by an S wave 7 mm. deep. A barely discernible initial downstoke was found in V_2, followed by an equiphasic RS complex, each component being approximately 12 mm. in

If the initial downstroke in V_1 and V_2 represented a Q wave, a diagnosis of old septal infarction would have been justified.[47] Comparison of measurements of the QRS interval in various precordial leads provides a useful method for determining whether or not the initial downstroke in V_1 and V_2 is a part of the ventricular complex. When differences exist.

the longest measurement is generally obtained in leads directly over the ventricles (such as V_3 and V_4) and slightly shorter measurements are obtained in leads at a distance, such as V_1 and/or V_6, owing to transmission losses at onset or end of ventricular activation. The interval from the beginning of the initial downstroke to the end of the S wave in V_1 measured 0.12 second and was 0.03 to 0.04 second longer than the interval in V_3 and V_4. This observation indicated that the initial downstroke was not a part of the QRS complex, but rather an antecedent P wave. The resemblance of the downstroke of this P wave to the exceptionally steep descent of the T wave suggested that the latter was due to a superimposed P wave. This was confirmed by the fact that the time interval between these two downstrokes was exactly one-half of a cycle length.

Although the correct diagnosis could be reached from a careful analysis of Lead V_1, it was evident from a glance at Lead V_{3R}. The inverted and notched deflection immediately preceding the RS complex was unmistakably a P wave and the presence of a similar notch on the T wave indicated a second superimposed auricular deflection. The shape of the P waves and the intervening isoelectric interval indicated the presence of auricular tachycardia and the A–V ratio indicated a 2:1 block. A similar rhythm was found in five other tracings obtained over a span of sixteen days. The ease of diagnosis from Lead V_{3R}, the possibility of mistaking the P for Q waves from a casual inspection of Leads V_1 and V_2 and of detecting the abnormal rhythm from a more careful analysis of the same lead, and the difficulty or impossibility of diagnosis from other leads were also observed in other tracings.

Death occurred as a result of multiple pulmonary infarcts. At autopsy the heart weighed 554 grams and showed right and left ventricular hypertrophy, associated with rheumatic mitral and aortic valvulitis. Both atria were markedly dilated. The coronary vessels were of normal caliber and no evidence of myocardial infarction was found.

Summary

Certain QRS-T patterns suggestive of myocardial infarction, but actually referable to alterations of blood potassium, to myocardial ischemia, to subepicardial myocarditis, or to distortion from arrhythmia are illustrated by cases in which the possibility of myocardial infarction was excluded at autopsy.

Precipitous fall in blood potassium to low levels may be manifested by RS-T depression and T-wave inversion resembling that associated with acute subendocardial infarction. Extreme hyperpotassemia may cause marked widening of the terminal portion of the QRS complex and fusion with the T wave to form a pattern that may be mistaken for that of myocardial infarction, complicated by bundle branch block.

The problem in differential diagnosis between acute subendocardial infarction and an acute surgical abdomen complicated by shock and the error that may result from overemphasis of electrocardiographic findings is exemplified by a case showing marked and progressive RS-T depression due to myocardial ischemia without infarction, secondary to gangrene of the ileum.

Rapidly evolving cove-plane inversion of the T waves associated with acute myocardial ischemia of varying cause and with acute subepicardial myocarditis is exemplified and differentiated from that accompanying acute myocardial infarction.

The following electrocardiographic patterns associated with subepicardial myocarditis and pericarditis may be mistaken for those of myocardial infarction and are differentiated with the aid of illustrative cases: (1) localized elevation of the RS-T junction and/or cove-plane inversion of the T wave in the presence of an initial R wave of normal or low voltage, (2) abnormal upward displacement of the RS-T segment and/or cove-plane inversion of the T wave in complexes exhibiting a distinct Q wave and a normal to borderline Q/R ratio, (3) localized reduction in the amplitude of the R wave in a given lead below that attained in adjacent leads to the right and left.

The errors that might result from attempts to draw diagnostic inferences from the registration of deep Q waves and/or cove-negative T waves in premature ventricular beats or in ventricular tachycardia are brought out by suitable examples. Furthermore, premature auricular beats may be conducted aberrantly through the uninfarcted ventricles, leading to the registration of abnormal QR patterns, simulating those recorded in sinoauricular beats in the presence of infarction. The fusion of a sharply inverted ectopic P wave in V_1 with the first portion of the QRS to produce a triphasic deflection resembling the findings in septal infarction is also demonstrated.

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Cinical Electrocardiography—Its Present Position and Possible Potentialities

By Louis N. Katz, M.D.

The present role of the electrocardiogram in clinical medicine is discussed and its limitations as a diagnostic tool are pointed out. Its particular usefulness in the analysis of arrhythmias and the value of certain specific empirically derived patterns are discussed. The misuse of the term "unipolar" is explained, the actual conditions present being only crude approximations of the assumptions upon which the equilateral triangle theory rests. The role of multiple V chest leads and the aV limb leads in clinical electrocardiography is outlined. A brief explanation of stereovectorcardiography and the great promise it holds for the future is presented.

IT IS A privilege to have been asked to give the Louis Gross Memorial Lecture. It was my good fortune to know Dr. Louis Gross, one of the most brilliant cardiologists, whose career unfortunately was cut short in his prime. It is an honor to be included with the distinguished lecturers who have preceded me.

Dr. Harold Segall asked me to discuss in this lecture the present position of clinical electrocardiography and its future potentialities. We are in a period of transition today just as in the 1930's. I recall a conversation with the late Sir Thomas Lewis in London in 1935 in which he informed me that he was giving up his studies on electrocardiography since "the cream had been skimmed off." This was just about the time when the brilliant work of F. N. Wilson and his associates and of C. C. Wolferth brought into clinical use the concept of chest electrocardiography which did much to revolutionize the clinical utility of the electrocardiogram. Are we in a similar situation today in regard to the aV limb and the V chest leads, so often loosely called "unipolar" leads, or is this a false tangent which is occupying the minds of those who find the empiric approach to clinical electrocardiography unexciting? Before considering this matter, however, I should like to summarize the traditional position today of the electrocardiogram in clinical practice as an empiric tool assisting the clinician in the diagnosis, prognosis, and management of his patients.[41]

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While I call this era an empiric one, this is not strictly correct because there is a rational background of theory concerning the factors contributing to the recording of the electrocardiogram which permits the intelligent clinician to make reasonable judgments about the source of the impulse, its manner of spread, the presence of injury effects and the derivation of several diagnostic criteria. This, however, does not nullify the fact that in the final analysis the electrocardiogram, like other clinical data, is interpreted empirically. It is as good as the judgment of the interpreter. If he lacks common sense, if he reads too much into the record, if he fails to correlate the evidence from this aid in relation to the other clinical evidence, if he uses this record to confirm the prejudices which he has reached before taking the record, then it would have been best had the electrocardiogram been omitted.

The position of the electrocardiogram in clinical medicine can best be expressed by a simple colloquialism "So your electrocardiogram is abnormal, so what!" It has been stated by C. B. Thomas that almost any kind of electrocardiogram may be normal in a person under 30 years of age. When a person reaches the age of three score and ten or more, almost any sort of electrocardiogram may be considered normal in the sense that change may be merely a part of the aging process. I wish to emphasize these facts because people forget that an isolated abnormal electrocardiogram does not inevitably indicate organic heart disease. It may imply some temporarily disturbed function such as in the innervation or hormonal control of the

heart, constituting part of the picture of psychosomatic disturbances—and this may actually have been engendered by fear or excitement associated with the taking of the record.

Even an abnormal electrocardiogram associated with organic heart disease may be benign or occult, requiring no alteration in the manner of living of the patient. The abnormal electrocardiogram is less important than the circumstances under which it occurs. Taking an electrocardiogram does not absolve the clinician from also using sound clinical judgment. Contrariwise, a normal electrocardiogram is no guarantee that the heart is normal. The electrocardiogram is only part of the clinical examination, an important adjuvant which should never be omitted in a cardiac patient or in a person in the coronary age group. Under such circumstances it serves as a scanning test possibly throwing out hints to the alert clinician to examine his patient more carefully in certain directions.[34, 47, 51]

Experience has shown that the clinical utility of the electrocardiogram depends upon the information it gives about several of the physiologic properties of the heart, including its irritability, rhythmicity and conductivity[2, 49]; it furnishes no information concerning the contractile power of the heart or its tone except that which experience has shown are associated with certain types of electrocardiographic changes.

The electrocardiogram is the final authority in unravelling the rhythms of the heart. It can tell where each impulse arises and how it spreads. Of course, as is true of the necropsy, the electrocardiogram may occasionally fail to give a completely satisfactory solution. For example, paroxysmal tachycardia occurs more often at the bedside than in electrocardiographic files because the attack may be over before the record is obtained. Sometimes the clinical history may be more helpful in distinguishing the nature of rapid heart action, as in differentiating sinus tachycardia and paroxysmal supraventricular tachycardia. Even with such limitations, the electrocardiogram can be considered the "living autopsy" for the diagnosis of cardiac arrhythmias.

It is important to diagnose the nature of arrhythmias. For example, the significance of each of the three commonest arrhythmias, sinus arrhythmia, premature systoles and auricular fibrillation, is quite different.[44] Sinus arrhythmia is a normal phenomenon although sometimes encountered in disease of the heart. Premature systoles occur occasionally in everyone, but when frequent, when from multiple foci, when causing bigeminal rhythm or occurring in pairs, they are usually associated with disease of the heart, with abnormalities of the patient or with some toxic drug action. Auricular fibrillation may occasionally occur in paroxysmal form in otherwise normal individuals but in most instances it is indicative of abnormality in the patient or disease of the heart. It is not always easy to distinguish between these and other less common arrhythmias at the bedside. Hence the electrocardiogram, the court of final appeal, is all important in this connection.

Certain conduction disturbances are almost exclusively an electrocardiographic diagnosis, viz. first degree A-V block, intraventricular block, electrical alternans and the Wolff-Parkinson-White syndrome.[44] The recognition of these conditions has important clinical implications. The electrocardiogram, therefore, has contributed to the clinical evaluation in this sense. One must recognize conduction disturbances which arise because of the normal refractoriness following stimulation which lasts about as long as systole and prevents stimulation of the involved heart area by another impulse coming at this time.[9] The normal refractory state may thus lead to interference, dissociation, aberrant conduction and even complex arrhythmias, all part of the normal physiology of the heart. These conditions must be sharply distinguished from disease of the heart that leads to prolongation of the refractory state, to abnormal depression of conduction and to block. Failure to distinguish between the normal and abnormal refractory phase and their consequences has led to improper reading of the electrocardiogram with resulting harm to the patient.

The second item of great value to be derived from the electrocardiogram depends on the analysis of its contour. Contour changes may

be non-specific or may fall into one or another specific pattern. [12, 25, 28-31, 34-37, 39, 40, 43, 44, 46-48, 51, 52, 58] These patterns, like specific clinical syndromes, have largely arisen on an empiric basis. One should recognize the more important patterns and their variations, and should realize that specific patterns are not always diagnostic of specific conditions since they may be imitated by unrelated circumstances. Contrariwise, the conditions which can give rise to specific patterns do not always do so. Thus, the proper use of electrocardiographic contour as a clinical aid depends upon broad experience, and by experience I do not mean the dogmatism of the expert who, over many years, has failed to recognize his errors. It is my firm conviction that the proper recognition of specific electrocardiographic patterns and the circumstances under which they occur will more than repay the clinician for the effort involved.

Before briefly discussing a few patterns, it is important to emphasize that the degree of electrocardiographic abnormality is not necessarily related to the amount of damage in the heart. The very nature of the heart's electrical properties permits opportunistic minor lesions to distort the electrocardiographic contour tremendously; in other instances more important and more extensive lesions may affect the electrocardiogram little if at all. The greatest value of the chest leads has been their ability to explore certain areas of the heart which have little or no effect on the ordinary limb leads. But even with all available leads one cannot quantitatively correlate the extent of abnormality in the heart with that in the electrocardiogram. Furthermore, it is difficult to judge the nature of the process affecting the heart except from certain specific patterns or to tell from the electrocardiogram alone whether the damage to the heart is permanent or evanescent, congenital or acquired, functional or organic, or whether it is hypoxic, ischemic, nutritive, inflammatory or degenerative. It is for this reason that I refuse to say that an abnormal electrocardiogram, per se, always indicates heart damage. The electrocardiogram can only provide the interpreter with the fact that, on the basis of experience, the record is within normal limits, probably within normal limits, on the borderline, probably abnormal, or definitely abnormal. The significance of this judgment, as far as damage to the heart is concerned, will depend upon the entire clinical survey.

Time does not permit an adequate discussion of the specific patterns, but I cannot resist the temptation to consider a few highlights.[44] The P-pulmonale pattern may occur in chronic cor pulmonale, or in perfectly normal hearts with a pendulous position. The mitral P wave is usually encountered in rheumatic mitral stenosis and is suggestive of impending auricular fibrillation, but may sometimes occur in other circumstances. The presence of the mitral P wave and of P-pulmonale helps to reveal that the causative lesion has led to dynamically important changes in the heart.

This contribution is even more significant in the case of ventricular strain. Thus, for example, there is considerable difference in finding hypertension associated with or without ventricular strain since in the former one can feel assured that the hypertension has caused clinically significant strain of the heart. Ofttimes, physical findings and x-ray findings may not be revealing when the electrocardiogram suggests that the hypertension has caused hypertensive heart disease.

In the case of valvular deformities, the electrocardiographic evidence of ventricular strain may distinguish those valvular diseases which are purely acoustic from the more serious ones which have led to dynamic disturbances causing strain of the heart. It is well known that in the case of valve regurgitation, other things being equal, the smaller the leak the noisier the murmur. Consequently, judgment of the seriousness of a valvular regurgitation based only on the murmur it creates may be deceptive.

Certain patterns are diagnostic, including the retrograde P pattern in A-V nodal rhythm, the mirror image dextrocardia pattern, the patterns indicative of acute diffuse pericarditis and of acute cor pulmonale, and the large diphasic QRS pattern seen in congenital heart disease. The importance of the electrocardiogram in the diagnosis of the variety of congenital heart disease present is gratifying in view of the growing value of surgery.

Of all the patterns, however, by far the most important is that of the coronary group. The basic cause of these QRS-T contours is the injury effects occurring during the various phases of the heart cycle. Persistence of some of these changes may be due to the opening of a "window," the result of necrosis of heart muscle with replacement by scar tissue. Depending upon the direction of change in various leads, one can distinguish between posterior, anterior, lateral and septal wall involvement, and their combinations. From the character of the time course in seriatim records, one can distinguish between the transitory, the protracted and the chronic types of coronary insufficiency. Transient coronary insufficiency gives rise to the clinical condition of angina pectoris. The protracted coronary insufficiency, which leads to acute myocardial infarction, in most instances is due to acute coronary occlusion, particularly thrombosis. Chronic coronary insufficiency, which may be benign and nonprogressive or more malignant and progressive in character, is most commonly due to coronary arteriosclerosis.

It is common knowledge that the physical examination often is unsatisfactory in coronary disease and too often the history also may be unreliable and colored by the patient's temperament and motives. The electrocardiogram has thus become so important in this common form of heart disease. The electrocardiogram has contributed to the earlier and more accurate diagnosis of coronary heart disease. In short, our ability to recognize the earlier and milder forms of coronary disease has changed our concepts of the range of severity of this group of diseases.

We have become so conscious of coronary disease that there is a tendency to read into a case evidence of coronary disease which may not be justified, and this error often is aggravated by similar misreading of the electrocardiogram. A rule that I have found extremely useful is that "not everything coronary *is* coronary." The contrary fact is also true, namely, that clinically significant coronary disease may be present when the electrocardiogram is normal or shows abnormalities of a nonspecific character.

It is because of this last circumstance that two tests have been developed as an adjuvant for clinical and electrocardiographic diagnosis, namely, the anoxemia and the exercise tests for coronary insufficiency.[13, 44, 50, 60] Earlier experiences with anoxemia have left me with the definite impression that it is much too hazardous a test to be used clinically, despite its ardent advocates. We employ the exercise test. In this test we have insisted that a physician perform it and only when the evidence clinically or electrocardiographically is inadequate for the diagnosis of coronary insufficiency, that the test be stopped whenever the patient develops pain or any other disability, that the records be taken not only immediately after the exercise but also a few minutes later, that the judgment of the test be based both upon the quantitation of the S-T deviations produced and upon the change in contour of the ST-T segment, and that the test be run with a metronome against definite standards. We have employed those standards established by Master upon his stair and we have used the double speed test. In our hands, this test has been of value in that a positive result has suggested coronary disease of clinical significance. A negative test, of course, does not exclude this possibility. We have not encountered any false positives even in cases of anxiety neurosis and the like, although others have claimed to have obtained them.

I believe that what I have stated so far should convince you that when properly employed the electrocardiogram has value in diagnosis and in prognosis. With a proper diagnosis the most important element for proper management is established. But the electrocardiogram may be of value also in following the management and therapy. From the residue of abnormalities remaining, as well as from other evidence, the amount of permanent damage after myocardial infarction and after acute rheumatic fever may be determined and management of the case thereafter planned accordingly. The electrocardiogram is useful in following the conversion of ectopic rhythms to sinus rhythm.

Digitalis is a commonly employed drug and a check upon its use may come from the elec-

trocardiogram not only by virtue of the characteristic digitalis pattern which it produces but also by its aid in establishing the character of the arrhythmias which digitalis intoxication may engender. Certain arrhythmias are signs of excess and are indications to stop or decrease its use, viz. frequent ventricular premature systoles of multiple origin, bigeminal rhythm, paroxysmal ventricular tachycardia, S-A block and advanced A-V heart block. This is not true of the digitalis ST-T contour with its associated abbreviated electrical systole, which may occur sometimes even with doses of digitalis below therapeutic levels. The digitalis contour should be considered a caution sign, not a stop sign.

The electrocardiogram may be of value in disclosing the inadvertent removal of too much parathyroid during thyroidectomy by a prolongation of the electrical systole. Certain characteristic T-wave changes and conduction disturbances may help to warn the clinician employing quinidine that he is beginning to tread on dangerous ground. The value of the electrocardiogram is established in determining high and low blood potassium and low blood calcium values in various conditions.

In summary, we find that the electrocardiogram is a valuable adjunct which cannot be overlooked, but its value depends upon its being read and correlated properly: neither underemphasized nor overstressed. It gives information not available from other clinical sources.

Unlike previous years, we cannot end the story at this point. A certain trend, a certain ferment, is in our midst which is upsetting even the most stable of us in this field. It had its beginnings even before the late war. With the advent of the war and the free employment of electrocardiography among masses of normal young people, the mass of normal data has broadened tremendously. So has the number of serious students in the field of electrocardiography, but many do not have the traditional background of my generation. As a consequence there has been a shift of emphasis in electrocardiography, and an urge among some who are poorly versed in mathematical theory and physics, to view the electrocardiogram as a method which, with certain assumptions, can precisely reveal facts based scientifically on physical laws of electricity. The consequence has been loose talk about unipolar leads which are really not unipolar and of elaborate mathematical formulation too often unsound, the assumptions which form their basis having been forgotten. Now we must stop and take stock. We must sift the grain from the chaff. There is much truth in the new developments but the revolution may not be as far reaching as some would have us believe. I should like briefly to review the theory of the electrocardiogram, to discuss the merits of the newer leads and to peer with you into the crystal of the future.

In considering the theory of the electrocardiogram, the subject naturally divides itself into two parts[2, 6, 23, 44, 49]: the creation of electric currents by the heart and the manner in which the currents spread through the body.

Currents are generated in the heart because the resting polarized state of its cell surfaces is destroyed nonsimultaneously when the heart is activated. If the heart were activated to an equal extent simultaneously throughout its mass, no electric currents would be generated. It is asynchronism of activation that gives rise to the electric currents. While interfaces within the heart muscle doubtless may be electrically polarized, and while other electrical properties such as resistance, impedance, capacitance and inductance are altered when the heart is stimulated, these facts may be ignored without serious error for the purposes in hand, and attention may be concentrated on the state of electrical polarization of the surface of the cells. Furthermore, whatever conclusions may be derived on histological grounds regarding the multiplicity of cells within the auricles and ventricles, the facts from the point of view of electric currents are most clearly revealed on the likely hypothesis that the heart consists of two cells, both syncytial: the one constituting the auricles, the other the ventricles.

These two complex syncytial cells do not completely enclose the cavity of the auricles or ventricles. They are incomplete due to the A-V junction. This is of importance in understanding the genesis of the electrocardiogram, since it leads to unbalancing of the electrical forces

which otherwise would not occur if the heart cavities were completely enclosed by these two syncytial cells. Were the latter the case and were the impulse to start in the exact center of each respective septum and spread at a uniform rate throughout each of the two cells and were the thickness of the outer walls the same, then no currents would appear outside the heart even though the stimulation were asynchronous. The fact that the floor of the auricles and the roof of the ventricles are free of muscle therefore is an important factor in the spread of the electrical currents to the body. So is the fact that the left ventricle has a thicker outer wall than the right.

The electrical activity of each of the two syncytial cells includes two asynchronous active processes: the one being stimulation, the other restitution. Stimulation is a period of depolarization which changes the two cells from a polarized to a depolarized state. Restitution is a period of repolarization which restores the depolarized cells to their polarized state. Two factors determine the asynchronism occurring during repolarization: (a) the topography of stimulation and (b) the unequal duration of electrical activity in the various fractions of the heart.

Empirically a normal relationship has been established between the topography of activation and that of restitution of the ventricles and this holds for changes in ST-T with various normal QRS contours. The relationship between the QRS and ST-T complexes constitutes the ventricular gradient which attempts to define the influence of activation upon recovery in a quantitative fashion so as to reveal whether changes in the ST-T complex are to be ascribed to alterations in the pattern of activation or occur independently of it. On the basis of experience, Ashman has determined the normal range of the ventricular gradient considered as a vector. A recent survey in my department has shown that the empirical correlation of restitution with activation of the ventricles, on the basis of inspection of the initial and final deflection of the electrocardiogram, coincided reasonably well with the more exact but more time-consuming analysis by means of calculation of the ventricular gradient.[59] In short, with experience one can obtain an idea of how much of the deformity of the final ventricular deflection is to be expected as the result of alteration in the spread of the impulse and how much is due to unrelated effects. Except as a check, from time to time, on the skill of the electrocardiographer I see no need to use the ventricular gradient in clinical electrocardiography.

As I have mentioned before, the heart, electrically speaking, consists of two complex syncytial cells. These are incompletely separated by the connective tissue in the A-V junction; the His-Tawara system constitutes the connecting link between them. In some instances there may be, in addition, a congenital anomaly variously located, consisting of a rapid bypass which results in the Wolff-Parkinson-White syndrome. The His-Tawara system is not neurogenic, it is muscular. It is not embryonic, it is neomorphic and specialized.

Besides the electric currents created by the asynchronous depolarization and repolarization of the auricular and the ventricular syncytial cells, two other causes exist for the development of electrical currents in the heart. The first is the after-potentials, such as occur in nerves, which explains the U wave.[38] The other is the result of injury effects.[15, 33, 49, 53] It is important to understand the genesis of injury effects since they form the basis of certain patterns, in particular of the coronary contours. There are a number of reasons for the development of injury effects. First, whenever part of the heart fails to respond because it is dead or replaced by scar tissue, it operates as if a new window had been created in the wall of the heart; the result being that the summation of the currents during activation and restitution are altered. Far more important, however, are actual injury effects that arise from the presence of injury to part of the living syncytial cell. These ensue under four sets of circumstances. First, the process of activation in the injured region may be retarded, causing an injury effect of depolarization which leads in the case of the ventricles to focal prolongation of the QRS complex. Second, the process of recovery may be retarded in the injured region causing an injury current of repolarization which leads in the case of the ventricles to the so-called characteristic coronary T wave with its peaked top, its symmetri-

cal limbs and its "soft shoulders." This is a common finding clinically and represents a mild form of injury. Third, the injured region may be partially or completely depolarized. This will give rise to injury currents of rest because when the uninjured region is fully polarized, the lack of this same state in the injured area will lead to potential differences; in the case of the ventricles it will result in a deviation of the T-P segment. Fourth, the injured region may be completely polarized but partially or completely unresponsive. This will give rise to injury currents of activity because when the uninjured region has been completely depolarized the unresponsive injured area will retain its electrical polarity, setting up potential differences; in the case of the ventricles it will cause deviations of the S-T segment. It is of course possible that various combinations of these four circumstances may occur simultaneously to varying degrees.

Finally, knowledge regarding the genesis of the electric currents by the heart requires some understanding, at least in broad terms, of the topography of the spread of the impulse through the heart. It is generally recognized that the impulse ordinarily arises in the sinus node and travels in general through the auricular syncytium to the left, caudad and ventrad, inscribing the P wave.

The situation in the case of the ventricular syncytium is more complex.[24, 44, 49] The impulse begins to invade the ventricular muscle in the septum, starting about one-fourth of the way down from the A-V junction, half way between the anterior and posterior interventricular grooves and closer to the left than the right ventricular cavity. From this starting point, it spreads radially in all directions. Being closer to the left than to the right cavity, it quickly reaches the former, so that the spread from left to right soon is not balanced by a spread from right to left. Next, the impulse wave front reaches the base of the septum at the A-V junction so that the spread towards the apex is not balanced by one towards the base. Thus in the first two stages, the unbalanced spread of the impulse front is at first from left to right across the septum and later down the septum from base to apex.

Eventually the impulse front reaches the anterior and posterior interventricular grooves somewhat nearer the base than the apex, and begins to spread from here simultaneously over the outer walls of the right and left ventricles in four wave fronts. These tend to neutralize each other except that they reach the base sooner than the apex, leaving the front moving towards the apex continuously unbalanced. Thus in the third stage, the unbalanced spread is down the outer walls from base to apex.

Two other factors begin to operate about this time and continue until stimulation is over, which further complicates the picture. The first factor is that in any given region of the outer wall of the ventricles, the subendocardium is activated before the subepicardium. There is, as a consequence, another direction to the wave front spread, one that radiates out from each ventricular cavity. The net effect of this extensive wave front spread would be an unbalanced spread up the outer walls from apex to base. This would at first neutralize the unbalanced spread toward the apex more and more, and would become the dominant direction of unbalanced spread as soon as the apical region of the heart was completely activated. The stimulation of the apex is completed before the base because the distance to the former from the starting point in the septum is shorter than that to parts of the base of the lateral wall of the ventricles. The second factor is that the left ventricle is thicker than the right. On the assumption of equal rate of spread, this would cause delay in the impulse reaching the outer surface of the left ventricle as compared to homologous points on the right ventricle. Actually the difference in arrival at homologous surface points is not too great because the impulse appears to spread out radially in the outer wall of the left ventricle and at an oblique angle pointed toward the base in the outer wall of the right ventricle. Because the wave front through the outer wall of the right ventricle spreads less to the right than the simultaneous wave front through the outer wall of the left ventricle spreads to the left, the spread to the left of the latter will give rise to a dominant leftward unbalanced element of spread of the wave front at this stage. In the fourth stage,

therefore, both of these factors would add an unbalanced spread over the outer walls of the ventricles from apex to base and from right to left. The QRS complex depends primarily upon the unbalanced wave fronts just outlined in the four stages of activation.

The genesis of the electric currents by the heart is only part of the picture. The rest comes from the fact that the heart lies within a solid conductor which has the shape of the torso, the four limbs and the head and neck having practically no influence. The flow of the electric currents generated by the heart through this solid conductor is complicated. I would like to develop the subject first in its simplest terms, as has been done by the Wilson school, by making certain assumptions, and then later to test their validity against the actual circumstances existing in the human body.[49]

If we assume that the torso is a sphere of homogeneous conducting ability of infinite size or at least of very large finite size, that the heart is in its center and is considerably smaller, approaching the equivalent of a point, then certain simple physical laws can be developed regarding the flow of electricity, as have been worked out by physicists. Furthermore, if we assume that the junctions with the torso of the three extremities, the left arm, right arm and left leg, form the apices of an equilateral triangle, then the laws that Einthoven enunciated would apply. Now both Einthoven and Wilson recognized the limits of these laws as applied to electrocardiography; their disciples have sometimes been less careful. In such a theoretical scheme, the impulse wave front can be synthesized from moment to moment in the heart cycle into a resultant three dimensional vector. This stereovectorcardiogram can be analyzed to determine the magnitude of the deflection obtained in the ordinary (Einthoven) limb leads and, for that matter, in any other lead. If this state of affairs existed and the proper indifferent electrode could be obtained, then truly unipolar leads could be recorded from the extremities and from the rest of the surface of the body.

Under such an idealized state, the exploring electrode could be considered as an eye looking at the miniature heart from one vantage point (fig. 1). The eye located in the left arm would view the translucent heart from a different aspect than one in the right arm, in the left leg, or in different places over the precordium. Under such circumstances, the law of the solid angle would hold precisely. The effect of each area of junction between polarized and depolarized regions of the syncytial cell—say that of the ventricles, and regardless of whether it were due to the depolarization process, to the repolarization process or to injury—would influence what the eye saw according to several laws. These would be: (1) the magnitude of the potential difference across the theoretical surface, (2) the orientation of the positive and negative poles on this theoretical surface, (3) the area enclosed by the theoretical surface's perimeter, (4) the inverse of the square of the distance from the center of the theoretical surface to the eye, and (5) the angle by which the eye was removed from the normal to the plane of the theoretical surface. The eye would synthesize what it saw of all the theoretical surfaces existing in the heart at the moment according to these laws, and reflect a simple image that we call the potential of the spot where the eye

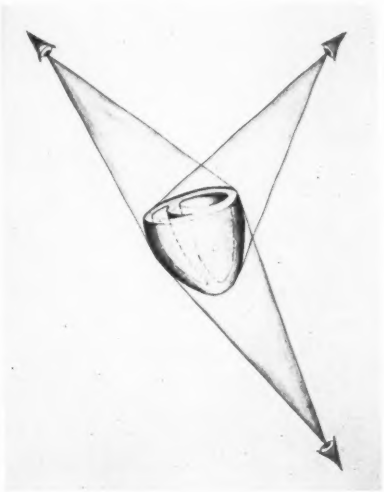

FIG. 1. Diagrammatic representation of the heart to show the "view" of the heart obtained by an eye placed in the right arm (upper left), left arm (upper right) and left leg (lower right). From the center of the pupil of each eye to the heart is shown the solid angle of the "view." Discussed in text.

is located. This is the principle of the unipolar lead and valuable information would be added by such leads were all the assumptions precisely true. It is obvious that "binocular" vision is a synthesis of two "monocular" images in the framework of experience. With inexperience, one might get confused in interpreting "binocular" vision.

To continue our analogy: unfortunately the medium between the hypothetical eye and the heart is not entirely transparent. Many factors contribute to cause distortion of the actual true image so that in the end the "monocular" view is like the image obtained by looking obliquely from above the surface of water at an object submerged beneath it, with the added complications that uneven waves are constantly rippling the surface and that the water is somewhat opaque. As all of you know, the visual image seen under these circumstances is a caricature of the real object below the surface of the water. What are some of these disturbing factors in the case of the heart's electric currents flowing in the body? First of all I must admit that practical experience shows that, despite their presence, some reasonable semblance of the true state of affairs can be obtained. The only thing that continues to annoy me is the failure of some protagonists to recognize the presence of these distortions and the tendency for them to accept the image which they see with their "monocular" leads as being the reality itself.

Reality is different from the ideal in several respects. First, the torso is not a large finite sphere but is ovoid or egg-shaped and not excessively larger than the heart. The eye in each extremity therefore gets a close view of the heart and finds it far from a point, and consequently all regions of the heart are not in one plane as theory demands. The eye is closer to certain parts than to others, and reports of the electrical architecture of the heart are thereby distorted.

Second, the solid conductor is not homogenous but, as work in my department has clearly shown, is heterogeneous.[4,14,20,21,23,44,49] Some parts conduct electricity well and others poorly, and this pertains especially to the solid conductor adjacent to the heart. For example, the lungs are poor conductors approaching a dielectric, and the liver and posterior muscle mass are excellent ones. As a consequence, what the eye will see will be distorted on this account. It will see those regions adjacent to good conductors better than those against poor ones.

Third, the heart is not in the center of the torso as the theory demands but is closer to the arms than to the legs and closer to the front of the chest than to the back. This too will alter what each eye will see.

Finally, the Einthoven standard three limbs used to record the electrocardiogram do not constitute, at their junction with the torso, an equilateral triangle: far from it. In some instances, as in the long and lanky individual, the triangle is more nearly a right angle triangle. Hence the Einthoven laws based on the equilateral triangle concept are only crude approximations.

Is it any wonder then that there should be a testing of all these laws against actual experience, and is it not fair to insist that the V leads and aV leads be known as such and not given the implication of exactitude which follows the use of the term unipolar for them? There are two ways by which the utility of these new leads may be ascertained. One is to test their actual degree of unipolarity, the other is to check their clinical value by the empiric approach.

It can be shown readily that when an electrode is placed *directly* on the exposed heart and connected to a distant electrode, one is dealing with a unipolar lead in the sense that the exploring electrode picks up so much more current from the heart than does the distant one that very little error arises in assuming that the distant one has no significant effect (fig. 2). This is particularly so for the recording of the electrical events directly under the exploring electrode—the so-called intrinsic effects. It is less true for electrical events elsewhere in the heart—the extrinsic effects. Under such circumstances, little difference is found regardless of which location is used for the distant electrode, and the difference obtained with such distant electrodes compared with the Wilson central terminal is practically negligible.

When however the ordinary precordial leads

are used, it can be shown that the location of the distant electrode exerts an influence because the exploring electrode is sufficiently removed from the heart. We have recently found, for example, that in one out of twenty cases the CF lead is sufficiently different from the V chest lead to cause a significant difference in clinical interpretation.[61] It is for that reason that we gave up CF leads in 1948 and are now employing exclusively the V chest leads, in which, as you know, the distant electrode is the central terminal of Wilson connected through five or ten thousand ohms to each of the three extremities: left arm, right arm and left leg (fig. 3).

This empiric judgment concerning the V chest leads was fortified by the results of a recent study in which we were able to show that, when men were immersed up to their mouths in a swimming pool, the Wilson central terminal in all of them was more nearly constant during the heart cycle than any other distant electrode.[62] However, we found that when the Wilson central terminal was compared with a central terminal of a large external triangle which could be assumed to approach a true null electrode, the Wilson terminal showed fluctations of some tenths of a millivolt during the heart cycle. Thus, the Wilson central terminal is not a null electrode but one that approaches this state much better and more consistently than the other distant electrodes routinely employed (fig. 2). Consequently, it is, on the basis of actual trial, more suited as a distant electrode for chest leads than an electrode on an arm, leg or the back.

The fact that the Wilson central terminal is not a null electrode makes the V limb leads non-unipolar. Since the overall deflection in the V limb leads are a matter of about 1 millivolt during the inscription of QRS and much less than this for the other deflections, it is apparent that errors of about 0.2 millivolt introduced by the central terminal of Wilson will distort the V limb leads significantly. This deviation can be readily demonstrated by algebraically adding the sum of V_L, V_R and V_F. Theoretically their sum should equal zero if they were truly unipolar leads in an equilateral triangle. Actually this is not true and often their sum is far removed from zero. It is thus not accurate to speak of them as unipolar leads. The use of the Goldberger modification,

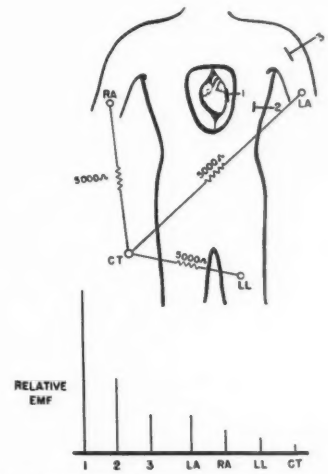

FIG. 2. Diagrammatic representation to illustrate relative electromotive force (shown in the diagram below) obtained by variously placed electrodes. *1* is an electrode directly on the exposed heart; *2* is one on the surface of the chest; *3* is one on the left arm. *RA*, *LA*, *LL* are electrodes on the right arm, left arm and left leg respectively. *C* is the central terminal of Wilson connected through 5000 ohms resistance to *RA*, *LA*, and *LL*. Discussed in text.

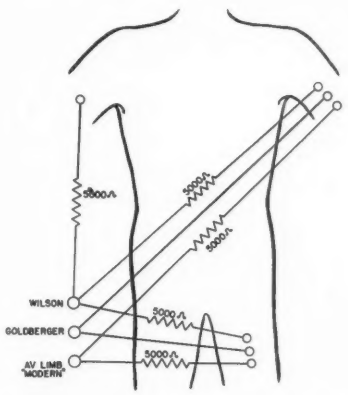

FIG. 3. Diagrammatic representation showing the Wilson central terminal, the Goldberger modification for aV limb leads and the "modern" aV limb lead connection which avoids some of the defects of the Goldberger modification. Discussed in text.

the aV limb leads, has merit in that the size of the deflections is increased one and a half fold,

but so is the influence of the distant lead. It has recently been shown, moreover, that the omission of the resistance in the connections from the limbs to the distant electrode, which Goldberger advocated, introduces another and significant error (fig. 3). Today it is recommended that when aV limb leads are taken the distant electrode have 5 to 10,000 ohms interposed between it and the two limbs connected to it.

To recapitulate, while any distant electrode may be practically useful when taking leads directly from the heart, the Wilson central terminal, though not perfect, is the best distant electrode to employ in the case of the chest leads. Even the Wilson central terminal, or the two-limb modification for augmentation, is far from an ideal electrode in the case of the V or aV limb leads. The V limb or aV limb leads are, therefore, much less unipolar than the chest V leads; so much so that it is, in my opinion, improper to call them unipolar leads. The merit of the aV and V limb leads must rest therefore on their practical utility.

Actual experience by Goldberger, Myers, and Sokolow, among others, has suggested certain definite clinical value for the aV limb leads. These we have recently been able to verify and amplify in our own experience in random sampling of over 1000 cases in the past two years. However, it must be pointed out that while extremely useful in these particular circumstances, additional information derived from aV limb leads not obtainable from the ordinary Einthoven limb leads or extensive chest lead exploration is found only in about 5 of every 100 cases. It would appear that the aV limb leads are particularly useful in the following circumstances:

(a) To help to distinguish between right heart strain and a normal right axis shift due to the position of the heart. The difficulty of diagnosing right heart strain in the electrocardiogram is apparent. The aid offered by the chest leads, when enough of them are taken, has become obvious; but even so the aV limb leads do assist in the judgment in a significant number of instances.

(b) High lateral wall infarcts of the left ventricle may be picked up in aV_L when other ordinary and chest leads may fail to reveal them. The only other valuable lead under these circumstances would be a precordial lead in the mid-axilla at the level of the second intercostal space.

(c) In an occasional instance, the aV limb leads may help to reveal whether a Q_3 is due to position or recent or remote myocardial infarction or coronary insufficiency. However, even here one must expect some disappointments as Pardee has just emphasized.

(d) In isolated cases, the aV limb leads may help to distinguish acute cor pulmonale from recent posterior wall infarction.

(e) The aV limb leads may reveal evidence of left heart strain, on occasion, before it is apparent in the V chest leads or ordinary limb leads.

(f) The aV limb leads may help to decide whether or not combined heart strain is present, and help to recognize left heart strain when the heart is vertical "electrically."

(g) Perhaps the most valuable utility of the aV limb leads has been in unraveling the influence of the wide range of positions of the heart on the contour of the electrocardiogram. The five electrical positions enunciated by the Wilson school, viz. the "electrically" horizontal heart, the semi-horizontal, the intermediate, the semi-vertical and the "electrically" vertical heart, are based on the appearance of the V or aV limb leads in relation to the V chest leads. This concept has helped to classify hearts electrocardiographically just as similar classifications have been useful in x-ray and fluoroscopy. The correlation of these "electrical" positions with the actual position of the heart revealed by x-ray and fluoroscopic examination has been good in many, but not in all, instances. The poorest correlation has been found when focal heart disease is present or when the heart is markedly enlarged, but occasionally the correlation is poor even in normal hearts. It is to meet these exceptions that Wilson introduced the adjective electrical. I myself would prefer to call these five combinations Wilson's combination I, II, III, IV and V since other influences besides position of the heart contribute to make these contours.

Let us consider this briefly. The heart has

three axes upon which it can shift (fig. 4). One is an axis running through the thorax from left to right which permits the heart's apex to shift ventrad or dorsad. Another axis runs through the chest from sternum to spine and permits the apex to shift to the right when the heart is pendulous or to the left when it is squat. The third is the long axis of the heart which runs from apex to base and permits the heart to rotate so that either the left or right ventricle becomes more ventrad. A composite of the position of the heart with respect to these three axes will help determine which of these Wilson contours the heart will approach, although the second is the most important.

Besides the resultant position just discussed and overall shifts of the heart, four other factors contribute in determining the electrical contour in the normal and therefore also in the abnormal heart.[49] The first is the nature and extent of the distribution of the good and poor electrical conductors in the conducting medium adjacent to the heart. The second is the shape of the heart (fig. 5): thus, the heart may be long and narrow or short and stocky with obvious alterations in the net direction of spread of the impulse in the ventricles. The third is the particular detailed anatomy of the conducting tissues within the ventricles. The fourth is the rate and sequence of the spread of the impulse. These four circumstances will contribute as much as the position of the heart in determining the electrical pattern in the aV limb leads and will explain the otherwise inexplicable deviations of the "electrical" from the anatomical position encountered in the normal heart.

Unfortunately, the conditions in which "electrical" position might be expected to be most valuable are often those in which it is most unreliable, namely the presence of injury currents, of focal conduction disturbances and of complex heart strains. At the present moment, the greatest divergence of opinion concerning the value of the "electrical" position of the heart is to be found under just such circumstances. I believe we must await the future to determine the extent to which we can rely upon so-called electrical position to account for the deviations seen from expected standard patterns. It is difficult to decide for the moment how much of these deviations from standard

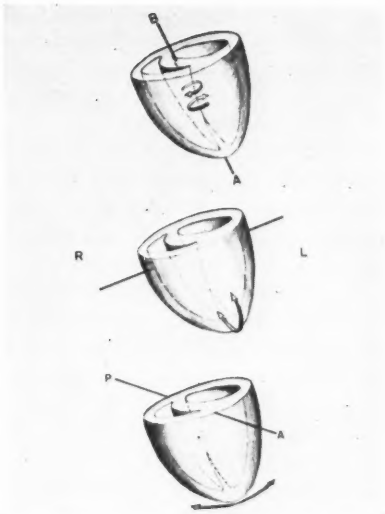

FIG. 4. Diagrammatic representation of the three axes upon which the heart can rotate. Top figure represents rotation on the long axes of the heart running from the base (*B*) to apex (*A*); the clockwise and counter-clockwise rotation is indicated by the arrows. Middle figure represents rotation on the axis of the heart running from right (*R*) to left (*L*); the arrows at the apex of the heart show the forward and backward movement of the apex possible on this axis. Lower figure represents rotation on the axis of the heart running from the spine (*P*) to sternum (*A*); the arrows at the apex of the heart show the leftward and rightward movement of the apex which make the heart squat or pendulous, respectively. Discussed in text.

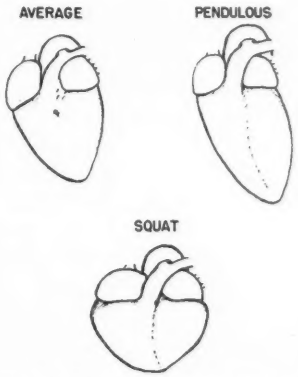

FIG. 5. Diagrammatic representation to show the variation in the shape of the normal heart (in exaggerated form).

patterns is caused by the combination of factors which contribute to the determination of "electrical" position and how much is due to complex combinations, such as bilateral bundle branch block, bundle branch block with focal intraventricular block, heart strain and focal intraventricular block, combined heart strain, focal injury currents combined with heart strain or with block in the bundle branch system, etc. For myself, I view with suspicion the glib assumption which some of the more enthusiastic advocates of aV limb electrocardiography make, that all or even most of the deviations from the expected in abnormal electrocardiograms are to be attributed to peculiarities in the so-called electrical position of the heart. That "electrical" position of the heart plays some role cannot be denied but the extent to which it does so remains yet to be determined by judicious empiric studies of these conditions.

While the aV limb leads have clarified our thinking in certain regards, they have, in their turn, created new dilemmas. This is to be expected with any modification in medicine. Old problems are solved, new ones are created.

The value of the V chest leads is definitely established. The only question that remains is how many and which chest leads should be taken. Previously, we had been taking V_2, V_4 and V_5, and judging their interpretation on empiric criteria. Our recent survey in which we explored the chest from V_{4R} to V_7 has convinced us that we should routinely use V_1 to V_6 and we have been doing this for some time. By using these six chest leads we are in a better position to estimate the presence of chronic or acute coronary disease, or recent and remote infarction, and to determine ventricular strain patterns. The omission of V_1 and V_6, we have found in our recent experience, seriously detracts from the value of the chest leads. With these six chest leads, one is also able to evaluate rotations of the heart on its long axis as well as to judge, with the assistance of the aV limb leads, other shifts in the heart's position.

Under special circumstances, it may be necessary to take other chest leads than these six. For example, it may be necessary to take a chest lead high up in the left mid-axilla, over the xiphoid, or over the sixth or seventh intercostal space in the left anterior or mid-axilla to help to detect small local myocardial infarcts. Sometimes it may be necessary in heart strain and in bundle branch system block to take V_7 or V_{5R} or V_{4R} for diagnostic purposes, when marked rotation or displacement of the heart is present. When auricular deflections need to be exaggerated and ventricular ones minimized, as in the diagnosis of auricular ectopic rhythm, it may be necessary to place electrodes in the second or third intercostal space over or at the margin of the sternum.

The clinical utilization of esophageal (E) leads may be questioned. Considering the amount of information to be procured as against the discomfort to the patient and the hazard of aggravating the cardiac status, particularly in the presence of coronary disease, I strongly oppose the ordinary clinical use of esophageal leads.

Recently considerable information has been obtained by the use of intracavity leads in which an exploring electrode has been placed in various positions within the cavities of the right or left heart in the experimental animal. More recently such intracavity leads have been used in man with the electrode in the cavity of the right heart, and even in the cavity of the left heart—a rather hazardous procedure. It is apparent without further ado that such intracavity leads are not for current clinical use and I doubt very much if they will ever become a routine procedure.

As I view the future of clinical electrocardiography, I can see in the immediate future a more precise and sharper definition of the exact value of these multiple newer leads in clinical practice, a judgment based on empiric correlation with established clinical cases and with necropsy checks. I can see the ardent protagonists and antagonists compromising their view until, in the not too distant future, universal agreement will be obtained. However, I must admit my own prejudice by stating categorically that the aV limb leads will never add as much to clinical electrocardiography in the sense that the introduction of the chest leads did.

As I view the future of clinical electrocardiography, I see more clarification of the various

patterns now recognized, with a more accurate definition of the sub-varieties, the pitfalls in their diagnosis, and the more precise assay of the circumstances giving rise to them. New patterns will doubtlessly be discovered to help enhance clinical electrocardiography. The most immediate advance I predict will come from the appreciation of combined heart strain patterns and of the varieties of patterns arising from congenital anomalies of the heart. The ability to define focal intraventricular block more precisely is also in the immediate future and its more common recognition will help to eliminate some puzzling contours. Of course, further clarification is in the offing regarding complex arrhythmias with the consequent advancement of our knowledge of the clinical physiology of the heart.

The "promised land" of the distant future is the stereovectorcardiogram. Already the beginnings of the exploration have begun. The theoretical background is established. Some of the limitations and practical difficulties are beginning to be understood. Let us see where we are going.

As mentioned earlier, the electric currents created by the heart can be considered as vectors. Whether dealing from moment to moment during the heart cycle with each particular fragment of the heart or with the heart as a whole, it must be recognized that electricity created in a solid conductor, which the body is, will have quantity, sense (positive to negative orientation) and direction. These are the attributes of a vector. The vectors of the various parts of the heart or the resultant of the entire heart will change constantly during the heart cycle. Since both the heart and the solid conductor are three dimensional objects, it follows that the changes in the vector during the heart cycle can be expressed in three dimensions, and this is the stereovectorcardiogram. The difficulties which remain are the practical ones of obtaining a good stereovectorcardiogram. They arise from the imperfections of the actual conditions of the heart and the body in contrast to a theoretical idealized scheme, as discussed above.

Two principles for obtaining a stereovectorcardiogram are possible. One is by the establishment of an equilateral tetrahedron with its apices on the body surface. The other is the establishment of a cube with its corners on the body surface. In either scheme, one can simultaneously take three leads along the three edges coming off from one corner of the solid constructed figure. These leads can then be registered at the same time upon two cathode ray oscillographs in which the electron beam passes three sets of electrodes 120 degrees apart. These oscillographs are modifications of those used in television. Each lead is connected simultaneously to a homologous pair of electrodes in each cathode tube. The electron beam striking the fluorescent surface of the cathode tubes would create a standing wave on each which could be photographed. By proper orientation of the electrode pairs in each cathode tube, the images from the two tubes would be sufficiently different so that when viewed in an old fashioned stereopticon, such as our parents used, the impression of three dimensions would be obtained. In this way three dimensional loops of the P wave, of the QRS complex and of the ST-T complex could be recorded which would express the fluctuations in direction and magnitude of these electrical events in the heart cycle. By means of a circuit periodically affecting the intensity of the electron beam, progressive increases in the intensity of the moving fluorescent image with momentary interruptions could be created. In this way the direction of the moment-to-moment shift of the stereopticon figure could be obtained from the arrow-like record, as well as the time sequence of inscription of each part of the loops from the interruptions. With this arrangement everything needed for analysis of an integrated stereopticon picture is available.

By establishing patterns of various conditions of normality and abnormality, for sundry positions of the heart, for heart strains, for intraventricular blocks and for the specific patterns, a series of standard pictures of the contour of the electrocardiogram could be synthesized for the clinician for comparison against the record of an unknown patient. He could compare the stereopticon obtained on his patient with the patterns established by experts based on experience and so in reality get the

benefits of the myriads of leads available without the necessity of actually using them all and learning their standards of normality and abnormality. Of course, experience will cause successive changes in the stereopticon standards employed until ultimately they become stereotyped.

One failing of this method is that small focal changes like focal intraventricular blocks and focal injury currents may be "washed out" in such an integration. However, these could quickly be revealed by employing a moving electrode exploring the entire chest and connected to a Wilson central terminal. That such a moving electrode is feasible was shown recently in certain animal experiments by Hellerstein and myself.[53]

The stereovectorcardiogram obtained would be a distorted picture of the stereovectorcardiogram that the heart actually creates. It is better so because it will prevent the ultra-mathematically minded from giving the stereovectorcardiogram more meaning than it has. No elaborate formulations involving trigonometry, calculus and the like will be required. No erudition will need to be employed. The clinician will simply learn the appearance of classical patterns, knowing full well that they are derived on an empiric basis, and will match the records of his patients against the standards which will be given him. Obviously in such a simplified system, the memorizing of large quantities of data so necessary today in clinical electrocardiography will not be required.

While I may seem to be facetious, actually I am serious in my belief that in stereovectorcardiography I see the handwriting of the future. Ordinary leads now so widely used will be employed only in special circumstances, as in the unraveling of the arrhythmias. Contour of the record will be integrated with the stereovectorcardiogram. Beginnings along these lines have already been made in North America and abroad.

There is still a long way to go. Until this goal is reached, the task of the clinician will be to use routinely the least number of electrocardiographic leads which will give him the maximum empiric information for the diagnosis, prognosis and management of his cases. In the use of multiple leads, as in other circumstances, the factor of diminishing returns applies. Finally, regardless of the number of leads employed or the theoretical background applied, ultimately the electrocardiogram is as good in clinical practice as the person who uses and reads it.

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Standardizing Factors in Electrocardiography

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Modifications in recorder standardization are required to correct inherent scalar differences among potential differences measured in a given electrocardiographic reference frame. In the frontal plane, agreement between the resultant vectors calculated from unipolar limb lead measurements and from standard lead measurements can be obtained by standardizing the galvanometer for recording the unipolar limb lead potentials at $\sqrt{3}$ cm. per millivolt. A method for calculating this "standardizing factor" and similar factors for other reference frames is presented and applied to calculations for the equilateral tetrahedron, the isosceles tetrahedron, and the cube.

INTRODUCTION

RECENT interest in spatial electrocardiography and vectorcardiography has resulted in the introduction of several spatial coordinate reference frames. A convenient method for correlating the data obtained in any such frame seems desirable. Need for such correlation in the frontal plane was indicated with the introduction of unipolar limb leads[1] when corresponding vectors determined from measurements with the unipolar limb leads and with the standard leads were found to differ in magnitude. To secure agreement, measurements from the unipolar limb leads had to be multiplied by the factor $\sqrt{3}$ or the galvanometer had to be standardized at $\sqrt{3}$ cm. per millivolt for recording unipolar limb lead potentials. As proved by Hill,[2] this "standardizing factor" $\sqrt{3}$ is required because the potential differences measured on the galvanometer are scalar quantities which are treated as vectors when added in the triaxial reference system. This difference between the sets of measurements is considered in Wilson's method of vectorcardiography in the frontal plane[3] when the sensitivity of the central terminal-to-foot circuit (a unipolar limb lead) is made $\sqrt{3}$ times that of the Lead I circuit. It is apparent that standardizing factors are required in spatial electrocardiography and vectorcardiography also.

In this paper two theorems are presented which facilitate calculation of standardizing factors for certain two- and three-dimensional frames whose boundary points represent electrode positions assumed to be *equidistant electrically* from the dipole of the heart. Application of these theorems to the calculation of standardizing factors for the Einthoven frontal plane and the equilateral tetrahedron is presented in detail.

THEOREMS

I. *The potential difference between two remote points coplanar with a dipole and equidistant from its center is proportional to the projected distance between these points on the axis of the dipole.*

Proof: Consider points A and B with coordinates x_1, y_1, and x_2, y_2, respectively, both at distance r from the center of the dipole. It is assumed that the dipole of moment D lies along the x axis with its center at 0 and that r is much greater than the distance between the dipole charges (fig. 1).

The following equations apply for the potentials produced by the dipole at points A and B:

$$\left.\begin{aligned} E_A &= \frac{KD}{r^2}\cos\theta_1 = \frac{KD}{r^2}\cdot\frac{x_1}{r} = \frac{KD}{r^3}\cdot x_1 \\ E_B &= \frac{KD}{r^2}\cos\theta_2 = \frac{KD}{r^2}\cdot\frac{x_2}{r} = \frac{KD}{r^3}\cdot x_2 \end{aligned}\right\} \quad (1)$$

where K is a proportionality constant. Therefore,

$$E_A - E_B = \frac{KD}{r^3}(x_1 - x_2) \quad (2)$$

where $(x_1 - x_2)$ is the projected distance between the two points on the axis of the dipole.

Discussion: If we consider two pairs of points $L(x_3, y_3)$ and $M(x_4, y_4)$, and $R(x_5, y_5)$ and $S(x_6, y_6)$

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all at distance r from the center of the dipole with $y_3 = y_4$ and $y_5 = y_6$, then L and M lie on one line parallel to the dipole and R and S lie on another line parallel to the dipole.

By application of theorem I,

$$E_L - E_M = E_{LM} = \frac{KD}{r^3}(x_3 - x_4) = \frac{KD}{r^3} \cdot \overline{LM} \qquad (3)$$

$$E_R - E_S = E_{RS} = \frac{KD}{r^3}(x_5 - x_6) = \frac{KD}{r^3} \cdot \overline{RS} \qquad (4)$$

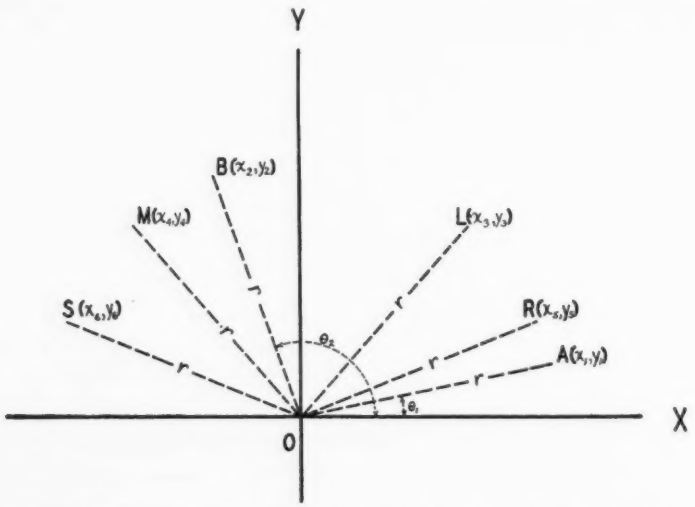

FIG. 1. Dipole at O and several points equidistant from O in plane XY.

where $\overline{LM}$ and $\overline{RS}$ are the distances between the respective points. Dividing equation 3 by equation 4, we obtain

$$E_{LM}/E_{RS} = \overline{LM}/\overline{RS} \qquad (5)$$

In this case the same dipole produced potential differences E_{LM} and E_{RS} between two pairs of equally remote points. However, if $\overline{LM}$ is greater than $\overline{RS}$, then E_{LM} is greater than E_{RS} by the ratio $\overline{LM}/\overline{RS}$. If E_{LM} is taken as the reference potential difference to determine the dipole moment, potential difference E_{RS} must be multiplied by the factor $\overline{LM}/\overline{RS}$ to make the measurements of potential difference alone indicate equal dipole moments. Consequently $\overline{LM}/\overline{RS}$ is the standardizing factor which must be applied to E_{RS}. Therefore, *the standardizing factor for potential differences between pairs of points equally remote from a dipole is inversely proportional to the distances between the points.*

(Note: For simplicity the preceding analysis was made in electrostatics. However, if the conducting medium to which the analysis is applied is electrically homogeneous and isotropic, the same ratios will apply to potential differences with current flowing.)

II. *The potential at the junction of* n *equal impedances connected to* n *points in space is the algebraic average of the potential of the* n *points.*

Proof: This is readily shown by application of an electrical network theorem of Millman.[4] Consider the circuit in figure 2.

The points *1, 2, 3, 4, . . . N* are at potentials $E_1, E_2, E_3, E_4, \ldots E_N$, respectively, with respect to the reference point 0. By Millman's theorem E_c, the potential of point C, the junction of the n impedances, is

$$E_c = \frac{\sum_{S=1}^{N} \frac{E_S}{Z_S}}{\sum_{S=1}^{N} \frac{1}{Z_S}} = \frac{\frac{E_1}{Z_1} + \frac{E_2}{Z_2} + \frac{E_3}{Z_3} + \cdots + \frac{E_N}{Z_N}}{\frac{1}{Z_1} + \frac{1}{Z_2} + \frac{1}{Z_3} + \cdots + \frac{1}{Z_N}} \qquad (6)$$

If

$$Z_1 = Z_2 = Z_3 = \cdots Z_N = Z$$

then

$$E_c = \frac{\frac{1}{Z}(E_1 + E_2 + E_3 + \cdots + E_N)}{\frac{N}{Z}} = \frac{E_1 + E_2 + E_3 + \cdots + E_N}{N} \qquad (7)$$

Therefore, E_c is the algebraic mean of the n potentials.

Discussion: This theorem is useful for those cases in which potential differences are measured between the Wilson central terminal or similar balanced central terminals and an exploring electrode. For the particular reference frame and central terminal configuration selected, it facilitates the calculation of the potential of the central terminal. However, if the impedances between the central terminal and the electrode positions on the body are unequal, the potential of the terminal is calculated by use of equation 6.

As a corollary of this theorem it should be noted that potential E_c cannot be greater than the maximum potential of points *1*, *2*, *3*, *4*, . . . *N*.

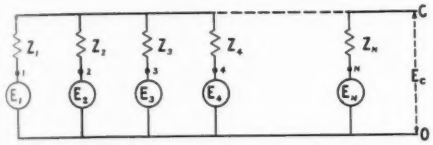

FIG. 2. Schematic diagram of a generalized central terminal derivation (Millman's theorem).

APPLICATIONS

In the applications considered in this paper the following are assumed:

1. The center of the dipole is electrically equidistant from the electrodes between which the potential differences are measured.

2. The medium in which the dipole is immersed is electrically homogeneous and isotropic and infinite in extent. These assumptions are common to the Einthoven concept of electrocardiography in the frontal plane and are here applied to spatial vectorcardiography also.

The mathematical procedure is simplified if the calculations for standardizing factor are based not on the ratio of the lengths of the vectors resulting from the different sets of measurements for a fixed dipole, but rather on the maximal potential differences which may be obtained in any one set if a given dipole is rotated in all directions about its center. The sets are chosen so that all maximal possible readings in the set are equal. From theorem I these maxima are obtained when the dipole is parallel to the line connecting the electrode positions in the coordinate frame.

I. *FRONTAL PLANE (Einthoven's equilateral triangle with Wilson's central terminal. Dipole at center of triangle. Figure 3)*

All resistors R_1 are equal. Therefore, from theorem I

$$e_C = \frac{e_R + e_L + e_F}{3}. \tag{8}$$

Since the dipole lies in the frontal plane with its center coinciding with that of the triangle,

$$e_R + e_L + e_F = 0^1. \tag{9}$$

Therefore,

$$e_C = 0. \tag{10}$$

In this case, the potential of the central terminal is zero. From the symmetric disposition of the equal positive and negative dipole

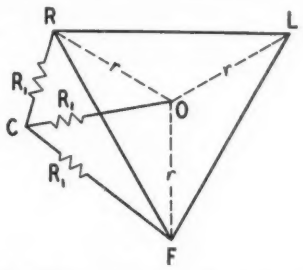

FIG. 3. Frontal plane with Wilson's central terminal.

charges about point O, the potential at point O is zero. The central terminal and the center of the dipole may be said to coincide electrically. If the dipole is parallel to RL, the potential difference between R and L, e_1, will be maximal, and by theorem I

$$e_{1_{max}} = \frac{KD}{r^3} \cdot RL \tag{11}$$

Similarly with the dipole parallel to FR,

$$e_{2_{max}} = \frac{KD}{r^3} \cdot FR \tag{12}$$

and with the dipole parallel to LF,

$$e_{3_{max}} = \frac{KD}{r^3} \cdot LF \tag{13}$$

Since the sides of an equilateral triangle are equal,

$$RL = LF = FR \tag{14}$$

and

$$e_{1_{max}} = e_{2_{max}} = e_{3_{max}}. \tag{15}$$

Therefore, if a dipole located at the center of the frontal plane is rotated in this plane, about its center, it will produce equal but, of course, not simultaneous maximal potential differences in each of the standard leads.

As shown, O and C are both at zero potential. Therefore, by theorem I, if the dipole is directed along OR

$$e_{OR_{max}} = e_{CR_{max}} = e_{R_{max}} = \frac{KD}{r^3} \cdot OR \qquad (16)$$

Similarly, if the dipole is directed along OL or along OF

$$e_{L_{max}} = \frac{KD}{r^3} \cdot OL \quad \text{or} \quad e_{F_{max}} = \frac{KD}{r^3} \cdot OF \qquad (17)$$

II. *EQUILATERAL TETRAHEDRON*

Case I. (Central terminal formed by joining equal resistors R_1 to the four apices of the tetrahedron. Dipole at center of tetrahedron. Fig. 4a). —From theorem I, the maximal potential differences between pairs of apices R, L, F, and B are

$$e_{RL_{max}} = \frac{KD}{r^3} \cdot RL; \qquad e_{LF_{max}} = \frac{KD}{r^3} \cdot LF; \text{ etc.} \qquad (19)$$

But *all* edges of the equilateral tetrahedron are equal, that is

$$\overline{RL} = LF = \overline{FR} = RB = LB = FB \qquad (20)$$

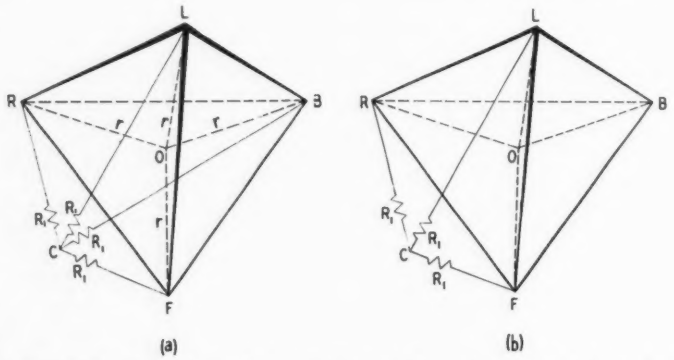

FIG. 4. (*a*) Case I. Equilateral tetrahedron with central terminal derived from electrodes R, L, F, and B. (*b*) Case II. Equilateral tetrahedron with Wilson's central terminal in frontal plane.

The relations among the medians and the sides of an equilateral triangle are

$$OR\sqrt{3} = OL\sqrt{3} = OF\sqrt{3} = RL = \overline{LF} = F\overline{R} \qquad (18)$$

Therefore,

$$e_{R_{max}}\sqrt{3} = e_{L_{max}}\sqrt{3} = e_{F_{max}}\sqrt{3} = e_{1_{max}} = e_{2_{max}} = e_{3_{max}}.$$

Consequently, although the maximal potential differences produced by a dipole as it rotates about the center of the frontal plane triangle are equal in the standard leads or in the unipolar limb leads, the maximal potential difference in the standard leads is greater by $\sqrt{3}$ than that of the limb leads. If the standard leads are taken as reference, then the standardizing factor to be applied to the unipolar lead measurements is $\sqrt{3} = 1.732$, which is the ratio of the length of the side to the median of the equilateral triangle.

Therefore,

$$e_{RL_{max}} = e_{LF_{max}} = e_{FR_{max}} = e_{RB_{max}} = e_{LB_{max}} = e_{FB_{max}} \qquad (21)$$

as the dipole is rotated in all directions about the center of the tetrahedron.

Similarly, the maximal potentials at the apices are

$$e_{R_{max}} = \frac{KD}{r^3} \cdot OR; \qquad e_{L_{max}} = \frac{KD}{r^3} \cdot OL;$$
$$e_{F_{max}} = \frac{KD}{r^3} \cdot \overline{OF}; \quad \text{and} \quad e_{B_{max}} = \frac{KD}{r^3} \cdot \overline{OB} \qquad (22)$$

But all medians of the equilateral tetrahedron are equal, or

$$\overline{OR} = \overline{OL} = \overline{OF} = \overline{OB} = r \qquad (23)$$

Therefore,

$$e_{R_{max}} = e_{L_{max}} = e_{F_{max}} = e_{B_{max}} \qquad (24)$$

Since points R, L, F, and B are symmetrically disposed with respect to the center of the dipole,

$$e_R + e_L + e_F + e_B = 0 \tag{25}$$

for any direction of the dipole. Moreover, the resistors connecting C to R, L, F, and B are equal. Consequently, by theorem II,

$$e_C = 0 \tag{26}$$

The central terminal in this case is at zero potential for any direction of the dipole and may be said to coincide electrically with the center of the dipole. Hence

$$e_{CR} = e_R; \quad e_{CL} = e_L; \quad e_{CF} = e_F; \quad e_{CB} = e_B. \tag{27}$$

As shown in appendix I,

$$\overline{OR}\left(2\sqrt{\frac{2}{3}}\right) = \overline{RL}, \cdots, \text{etc.} \tag{28}$$

Therefore,

$$\begin{aligned} e_{CR_{max}}\left(2\sqrt{\frac{2}{3}}\right) &= e_{CL_{max}}\left(2\sqrt{\frac{2}{3}}\right) \\ &= e_{CF_{max}}\left(2\sqrt{\frac{2}{3}}\right) = e_{CB_{max}}\left(2\sqrt{\frac{2}{3}}\right) \\ &= e_{RL_{max}} = e_{LF_{max}} = e_{FR_{max}} \\ &= e_{RB_{max}} = e_{LB_{max}} = e_{FB_{max}} \end{aligned} \tag{29}$$

Consequently, if the measurements of potential differences between the apices of the tetrahedron, corresponding to four electrode positions, are taken as reference, the standardizing factor to be applied to the unipolar lead measurements is $2\sqrt{\frac{2}{3}} = 1.627$.

Case II. (*Wilson's central terminal in the frontal plane. Dipole at the center of the tetrahedron. Fig. 4b*).—The same considerations as in case I apply to measurements between apices (between limb electrodes or between limb and back electrodes), but the standardizing factors for measurements from the frontal plane central terminal to the limbs and back must be determined.

The maximal potential difference between the central terminal and the back is obtained when the dipole lies along the median $\overline{OB}$ to the back. In this position the dipole is symmetrically located with respect to points R, L, and F, that is, the distances between these points and the center of the dipole are equal, and the angles between the medians connecting these points to the dipole and the axis of the dipole are equal. Therefore,

$$e_R = e_L = e_F. \tag{30}$$

Since terminal C is connected to R, L, and F by three equal resistors, R_1,

$$e_C = \frac{e_R + e_L + e_F}{3} = e_R. \tag{31}$$

With the dipole in this position, according to theorem I,

$$e_{RB} = \frac{KD}{r^3}\cdot\overline{AB}, \tag{32}$$

where A is the midpoint of the frontal plane and $\overline{AB}$ is the projection of $\overline{RB}$ on the axis of the dipole. But C and R are at the same potential; therefore,

$$e_{CB_{max}} = e_{RB} = \frac{KD}{r^3}\cdot\overline{AB}. \tag{33}$$

Maximal potential difference between R and B is obtained when the dipole is parallel to $\overline{RB}$. As shown in appendix I,

$$\overline{AB}\sqrt{\frac{3}{2}} = \overline{RB}. \tag{34}$$

Therefore,

$$e_{CB_{max}}\sqrt{\frac{3}{2}} = e_{RB_{max}} = e_{RL_{max}} = e_{LF_{max}} = e_{FR_{max}}, \tag{35}$$

etc.

and the standardizing factor to be applied to the central terminal-to-back measurements is $\sqrt{\frac{3}{2}} = 1.225$.

Now consider the potential differences between the central terminal and R, L, or F (the limb electrodes). The maximal potential difference between the central terminal and R will be obtained when the dipole is parallel to median $\overline{AR}$ in the frontal plane. Under this condition, by theorem I,

$$e_R = \frac{KD}{r^3}\cdot\overline{AR}, \tag{36}$$

where $\overline{AR}$ is the projection of $\overline{OR}$ onto the axis of the dipole.

Moreover, with the dipole parallel to the frontal plane,

$$e_R + e_L + e_F = 0 \tag{37}$$

and by theorem II,

$$e_C = 0. \tag{38}$$

Therefore, $$e_{CR_{max}} = \frac{KD}{r^3} \cdot \overline{AR}. \tag{39}$$

Similarly

$$e_{CL_{max}} = \frac{KD}{r^3} \cdot \overline{AL} \quad \text{and} \quad e_{CF_{max}} = \frac{KD}{r^3} \cdot \overline{AF}. \tag{40}$$

Since $\overline{AR}$, $\overline{AL}$, and $\overline{AF}$ are medians of the frontal plane triangle,

$$\overline{AR}\sqrt{3} = \overline{AL}\sqrt{3} = \overline{AF}\sqrt{3} = \overline{RL} = \overline{LF} = \overline{FR} = \overline{RB} = \overline{LB} = \overline{FB} \tag{41}$$

Therefore,

$$e_{CR_{max}}\sqrt{3} = e_{CL_{max}}\sqrt{3} = e_{CF_{max}}\sqrt{3} = e_{RL_{max}} = e_{LF_{max}} = e_{FR_{max}}, \text{etc.} \tag{42}$$

and the standardizing factor to be applied to the central terminal-to-limb measurements is $\sqrt{3} = 1.732$, as in Einthoven frontal plane measurements where the dipole is assumed to lie in the frontal plane.

The isosceles tetrahedron* and the cube shown in figures 5 and 6 respectively have been analyzed similarly. For reference the standardizing factors for all frames are tabulated in table 1.

With the isosceles tetrahedron, it is assumed that the Wilson frontal plane central terminal is used and that the dipole lies at the center of this frontal plane equidistant from apices R, L, F, and B. With the cube, it is assumed that the central terminal is the junction of the equal resistors connecting the eight corners, and that the dipole lies at the center of the cube.

Discussion

Throughout this study the calculation of standardizing factors has been associated with the potential and the apparent location of the central terminal in the coordinate frame. For visualizing the apparent location we have found it convenient to think in terms of the following model: Each resistor in the electrical system is replaced by a rubber band. All bands have the same elasticity and cross section but their unstretched lengths are proportional to the resistances they replace. The bands are joined at one end to form the central terminal and the other ends are fastened to the correct apical points of the coordinate frame. As the bands stretch, the position of the junction corresponds to the apparent location of the central terminal.

This concept of the central terminal must be regarded with care, because the apparent location of the central terminal cannot be used generally to calculate the potential of the terminal. Consider, for example, the central terminal in case I of the analysis of the equi-

Table 1.—*Standardizing Factors for Potential Differences in Several Reference Frames*

Einthoven Triangle Fig. 3		Equilateral Tetrahedron Case I. Fig. 4a		Equilateral Tetrahedron Case II. Fig. 4b		Isosceles Tetrahedron Fig. 5		Cube Fig. 6	
Potential Difference	Standardizing Factor	Potential Difference	Standardizing Factor	Potential Difference	Standardizing Factor	Potential Difference	Standardizing Factor	Potential Difference	Standardizing Factor
$e_{RL} = e_1$	1	e_{RL}	1	e_{RL}	1	e_{RL}	1	e_{PQ}, e_{PR}	1
$e_{FR} = e_2$	1	e_{FR}	1	e_{FR}	1	e_{FR}	1	e_{PT}, e_{QS},	1
$e_{LF} = e_3$	1	e_{LF}	1	e_{LF}	1	e_{LF}	1	e_{QU}, e_{RS},	1
e_R	$\sqrt{3}$	e_{RB}	1	e_{RB}	1	e_{RB}	$\sqrt{\frac{3}{2}}$	e_{RU}, e_{SW},	1
e_L	$\sqrt{3}$	e_{LB}	1	e_{LB}	1	e_{LB}	$\sqrt{\frac{3}{2}}$	e_{TU}, e_{TV},	1
e_F	$\sqrt{3}$	e_{FB}	1	e_{FB}	1	e_{FB}	$\sqrt{\frac{3}{2}}$	e_{UW}, e_{VW}	1
		e_R	$2\sqrt{\frac{2}{3}}$	e_{CR}	$\sqrt{3}$	e_R	$\sqrt{3}$	e_P, e_Q,	$2/\sqrt{3}$
		e_L	$2\sqrt{\frac{2}{3}}$	e_{CL}	$\sqrt{3}$	e_L	$\sqrt{3}$	e_R, e_S	$2/\sqrt{3}$
		e_F	$2\sqrt{\frac{2}{3}}$	e_{CF}	$\sqrt{3}$	e_F	$\sqrt{3}$	e_T, e_U,	$2/\sqrt{3}$
		e_B	$2\sqrt{\frac{2}{3}}$	e_{CB}	$\sqrt{\frac{3}{2}}$	e_B	$\sqrt{3}$	e_V, e_W	$2/\sqrt{3}$

* The name isosceles tetrahedron has been applied to one whose frontal plane (apices R, L, and F) is equilateral and whose frontal plane medians are equal to RB, LB, and FB.

lateral tetrahedron. From our model the central terminal coincides with the center of the dipole at the center of the tetrahedron and its potential is zero, as is correct. However, if the dipole were directed along one of the medians

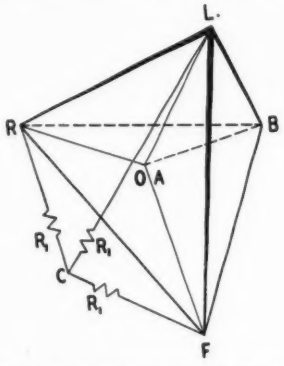

FIG. 5. Isosceles tetrahedron with Wilson's central terminal in frontal plane.

and the resistor along that median were changed slightly, the position of the central terminal would be considered to move along that median and no longer to coincide with the dipole. If the potential of the central terminal were calculated by the inverse square

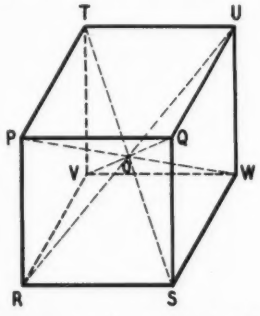

FIG. 6. Reference system employing the cube.

law from the distance between the central terminal and the center of the dipole, a large but incorrect potential would result. As noted in the discussion of theorem I, the potential of the central terminal can be no higher than the highest potential of the apices. Consequently, the maximal possible potential of the central terminal would be KD/r^2, which would occur only if the resistor were made zero so that the central terminal would be shifted to one of the apices. The potential of the central terminal is correctly determined in any case by application of Millman's theorem, equation 6. However, no error is made if the distances between this apparent position of the central terminal and the apices are used with theorem II to calculate the standardizing factors for the central terminal-to-apex potential differences.

A similar fallacy would arise in case II of the equilateral tetrahedron if the potential of the central terminal were determined from its apparent position at the center of the frontal plane and the inverse square law. No difficulty is encountered, however, if theorems I and II are employed.

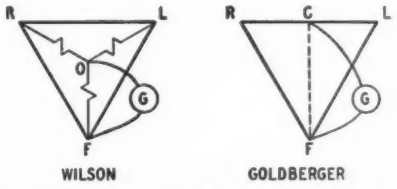

FIG. 7. Comparison of Wilson's limb lead with Goldberger's augmented limb lead.

If the center of the dipole and the apparent location of the central terminal do not coincide, the potential of the central terminal is not zero unless the dipole is perpendicular to the line connecting its center with the apparent location of the central terminal. A dipole located in front of or behind the frontal plane will, unless it is parallel to the frontal plane, produce a central terminal potential determined by the orientation of the dipole with respect to the apices of the frontal plane.

Application of the model and theorem I visually explains why Goldberger's augmented limb leads produce potential differences which are 1.5 times as great as those of Wilson's limb leads. In Goldberger's method the reference terminal is apparently shifted to the midpoint of one of the sides of the Einthoven triangle. Measurement of limb potential between this point and the limb involves a distance which is 1.5 times as large as that involved with Wilson's central terminal. This is shown in figure 7. The accuracy of Goldberger's method depends on equality of electrode-to-skin resistances. The central terminal is shifted

toward the electrode with the lower resistance. This shift can produce magnitude and polarity changes in some cases. The use of the 5000 ohm resistors in Wilson's method masks differences in electrode-to-skin resistances and reduces the magnitude of any error.

When the model for determining the apparent location of the central terminal is considered, the question might be asked: For determination of the limb potentials with a galvanometer with the use of Wilson's central terminal, does not the resistance of the galvanometer shunt one of the three equal resistors and thereby move the apparent location of the central terminal toward the limb being measured? The answer is: It does. Fortunately, however, the method of calibrating the galvanometer corrects any error. This is shown by applying Thevenin's theorem of electric circuits. Figure 8a is a schematic arrangement of the apparatus and the Einthoven triangle for determining the potential of the left arm by Wilson's method. Figure 8b is simply a rearrangement of figure 8a with the limb potentials e_R, e_L, and e_F made evident. If the galvanometer is disconnected, the network inside the dotted lines is open-circuited, and C and O are at the same potential. Therefore, the open-circuit output voltage, e_{CL}, in this case is e_L. The internal impedance of the network measured from terminals C and L into the box with e_R, e_L, and e_F short-circuited is $\frac{R}{3}$. The equivalent Thevenin network of the circuit within the dotted lines is shown in figure 8c. Since both e_L and the calibrating voltage e are in series with R_g and $\frac{R}{3}$, the method of calibrating the galvanometer compares e_L and the calibrating voltage directly. Therefore, the calibrated galvanometer indication is correctly e_L.

The effect of skin-electrode resistances was mentioned in connection with the possibility of errors in the use of Goldberger's augmented limb leads. This skin-electrode resistance also introduces differences between limb-limb potentials with and without the Wilson central terminal circuit. As long as the body may be considered as a homogeneous and linear conductor, the galvanometer, when used and calibrated in the standard way, records the potential difference which existed between the electrodes before the galvanometer was connected to these terminals. This was shown in connection with the study involving figure 8. Therefore, if the skin-electrode resistance is appreciable, it must be expected that IR voltage drops through these resistances when the Wilson central terminal network is applied will change the limb-limb electrode potential differences and likewise the central

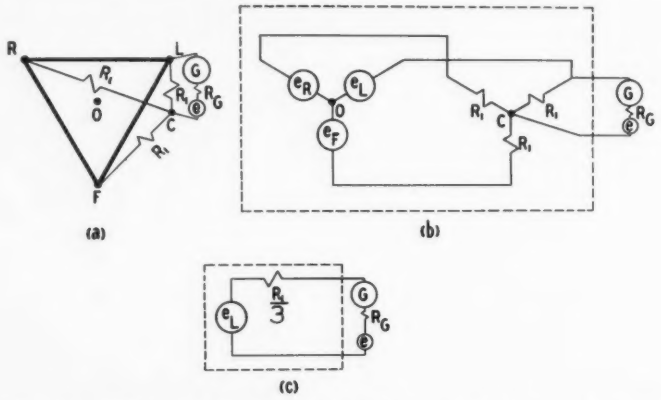

FIG. 8. Application of Thevenin's theorem of electrical networks to prove the accuracy of string galvanometer recordings of limb potentials. (*a*) Actual circuit arrangement. (*b*) Equivalent circuit arrangement. (*c*) Equivalent circuit arrangement according to Thevenin's theorem.

terminal-to-limb electrode potential differences. Thus, if the skin–electrode resistances are all 1000 ohms and the individual Wilson network resistors are 5000 ohms, the central terminal-to-limb potential differences as measured will be approximately 17 per cent less than they actually are, that is, the 1000 and 5000 ohms resistances are acting as a voltage divider to reduce the voltage presented to the galvanometer by $\frac{1000}{1000 + 5000} = \frac{1}{6} \simeq 17\%$. If the skin–electrode resistances are unequal, then a shift in the apparent position of the central terminal will occur which may introduce further errors. By careful application of the electrodes it is usually possible to reduce this error until it is within usual experimental electrocardiographic errors. However, in certain cases where the subject has very dry and thick skin, the error may be relatively large.

No attempt has been made in this paper to justify the validity of any coordinate frame for electrocardiography. Einthoven's frontal plane concept has been the basis of two-dimensional electrocardiography. As yet there is no standardization of frames for spatial electrocardiography and vectorcardiography. At present one of the tetrahedrons seems most suitable but there is little difference between results obtained with the isosceles and the equilateral tetrahedron concepts. The errors inherent in the use of any coordinate frame for electrocardiography must be realized. The body is neither infinite in extent, nor electrically homogeneous and isotropic. The electrodes are not necessarily so far removed from the distributed charges of the active cardiac muscle that these charges can be represented accurately by a dipole. However, such errors are probably well within the range of variation due to normal physiologic differences, as the work of Wilson and others regarding the validity of the Einthoven concept seems to indicate.

Summary

1. Standardizing factors are necessary to correlate measurements in any given coordinate reference frame employed in electrocardiography.

2. Two theorems are presented which facilitate the calculation of standardizing factors.

3. The application of these theorems to the Einthoven triangle and to the equilateral tetrahedron for spatial electro- and vectorcardiography has been shown in detail.

4. Standardizing factors for the Einthoven triangle, the equilateral tetrahedron, the isosceles tetrahedron, and the cube have been tabulated.

5. A concept regarding the apparent location of the central terminal has been presented, and with the theorems, discussed with respect to some applications in electrocardiography.

Addendum

Just before this paper was submitted for publication, a similar paper, "Le Problème des Derivations Bipolaires Appliqué à la Vectocardiographie," by E. C. Cabrera appeared in Acta Cardiologica **4**: 231, 1949. Whereas in our paper standardizing factors are based on the distance between the electrode locations projected onto the axis of the dipole, in Cabrera's paper they are based on the sine of half the angle between the lines connecting the center of the dipole with the electrode positions. Mathematically, for equidistant electrodes the two methods are equivalent. In our paper, however, we consider particularly the tetrahedron systems which have been used in the vectorcardiographic investigations of this laboratory and similar systems in which derived electrodes (central terminals) as well as direct contact electrodes are used.

Appendix I. Equilateral Tetrahedron

Consider the sagittal section of the equilateral tetrahedron shown in figure 9. This section is obtained by passing a plane through B and F perpendicular to the frontal plane. Points O, the center of the tetrahedron, and A, the center of the frontal plane, lie in the plane of this section. In the equilateral tetrahedron

$$\overline{RL} = \overline{LF} = \overline{FR} = \overline{RB} = \overline{LB} = \overline{FB}$$

and

$$\overline{OR} = \overline{OL} = \overline{OF} = \overline{OB} = r$$

In the section,

$$\cos \alpha = \frac{\frac{1}{2}\overline{FB}}{\overline{OB}} = \frac{\overline{AB}}{\overline{FB}} = \frac{\sqrt{\overline{FB}^2 - \overline{AF}^2}}{\overline{FB}}$$

Since $\overline{AF}$ is the median of the equilateral frontal plane

$$\overline{AF} = \frac{\overline{RL}}{\sqrt{3}} = \frac{\overline{FB}}{\sqrt{3}}$$

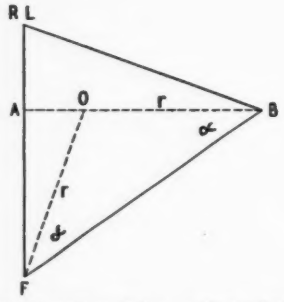

FIG. 9. Sagittal section of the equilateral tetrahedron.

Therefore,

$$\frac{\frac{1}{2}\overline{FB}}{\overline{OB}} \frac{\sqrt{\overline{FB}^2 - \left(\frac{\overline{FB}}{\sqrt{3}}\right)^2}}{\overline{FB}} = \sqrt{\frac{2}{3}}$$

or

$$\overline{OB}\left(2\sqrt{\frac{2}{3}}\right) = \overline{OL}\left(2\sqrt{\frac{2}{3}}\right) = \overline{OF}\left(2\sqrt{\frac{2}{3}}\right)$$
$$= \overline{OB}\left(2\sqrt{\frac{2}{3}}\right)$$
$$= \overline{RL} = \overline{LF} = \overline{FR} = \overline{RB} = \overline{LB} = \overline{FB}$$

Likewise

$$\overline{AB} = \overline{FB}\sqrt{\frac{2}{3}}$$

$$\overline{AB}\sqrt{\frac{3}{2}} = \overline{FB} = \overline{RL} = \overline{LF} = \overline{FR} = \overline{RB} = \overline{LB}.$$

APPENDIX II. Isosceles Tetrahedron

Consider the sagittal section of the isosceles tetrahedron shown in fig. 10. This section is obtained by passing a plane through B and F perpendicular to the frontal plane. Point O, the center of the frontal plane, lies in the plane of this section. In the isosceles tetrahedron

$$\overline{RL} = \overline{LF} = \overline{FR}, \qquad \overline{OR} = \overline{OL} = \overline{OF} = \overline{OB} = r,$$

and

$$\overline{RB} = \overline{LB} = \overline{FB}$$

Since the frontal plane is an equilateral triangle with medians $\overline{OF}$, $\overline{OR}$, and $\overline{OL}$.

$$\overline{OR}\sqrt{3} = \overline{OL}\sqrt{3} = \overline{OF}\sqrt{3} = \overline{OB}\sqrt{3}$$
$$= \overline{RL} = \overline{LF} = \overline{FR}.$$

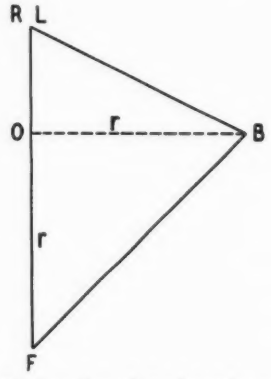

FIG. 10. Sagittal section of the isosceles tetrahedron.

In the triangle OFB of the sagittal section

$$\overline{FB} = \sqrt{\overline{OF}^2 + \overline{OB}^2} = \sqrt{\left(\frac{\overline{RL}}{\sqrt{3}}\right)^2 \left(\frac{\overline{RL}}{\sqrt{3}}\right)^2}$$
$$= \overline{RL}\sqrt{\frac{2}{3}}.$$

Therefore,

$$\overline{FB}\sqrt{\frac{3}{2}} = \overline{LB}\sqrt{\frac{3}{2}} = \overline{RB}\sqrt{\frac{3}{2}} = \overline{RL} = \overline{LF} = \overline{FR}.$$

APPENDIX III. Cube

In the case of the cube shown in fig. 6

$$\overline{PQ} = \overline{PR} = \overline{QS} = \overline{RS} = \overline{TU} = \overline{UW} = \overline{WV}$$
$$= \overline{TV} = \overline{PT} = \overline{UQ} = \overline{WS} = \overline{VR}$$

and

$$\overline{OP} = \overline{OQ} = \overline{OR} = \overline{OS} = \overline{OT} = \overline{OV} = \overline{OU} = \overline{OW}$$

But

$$\overline{UR} = 2\overline{OR} = 2\overline{OU} = \sqrt{\overline{UQ}^2 + \overline{QS}^2 + \overline{RS}^2}$$

Therefore,

$$2\overline{OR} = \overline{RS}\sqrt{3}$$

or

$$\frac{2}{\sqrt{3}}\overline{OR} = \overline{RS}, \text{ etc.}$$

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The Validity of the Equilateral Tetrahedron as a Spatial Reference System

By J. A. Abildskov, M.D., G. E. Burch, M.D., and J. A. Cronvich, M.S.

The error involved in the use of the equilateral tetrahedron reference system for electrocardiographic or vectorcardiographic studies has been investigated by means of an electrical dipole placed in the esophagus. Vectors plotted from the potential differences between electrodes defining the reference system were found to correspond fairly closely to the actual position of the dipole, indicating that the error is small.

THE STUDY of spatial vectorcardiography in progress in this laboratory prompted an examination of the equilateral tetrahedron, which is the reference system employed. In this system the shoulders, the pubis, and a point on the back represent the apices of the figure, and the assumption of the Einthoven hypothesis that shoulders and pubis are sufficiently removed electrically from the heart to be considered equidistant must also be applied to the electrode position on the back. On a purely anatomic basis, this seems justified, since in a study of 30 normal subjects this point on the back was found to be of equal distance from the heart as the left shoulder or even more distant.[1] It must also be assumed, as in the Einthoven hypothesis, that the body acts as an essentially homogeneous conducting medium.

It is generally agreed that these assumptions are approximations only, but are useful in frontal plane electrocardiography. If they can be applied with similar accuracy to a volumetric reference system, three dimensional vectorcardiography may provide new and valuable information.

In the report suggesting the use of the tetrahedron reference system, Wilson and coworkers[2] presented the results of an experiment designed to test its validity. An electric field was produced in a cadaver by means of electrodes inserted in various positions in the region of the heart. The potentials at the points representing the apices of the tetrahedron were then compared with those predicted on the basis of the known dipole position. As the authors pointed out, the results were inconclusive because of the special conditions of the experiment. The effect of death on tissue resistance and the effects of accumulation of fluid in the dependent portions of the body made the data difficult to interpret.

In the experiments reported herein, the potential differences between limb electrodes and limb and back electrodes produced by a "dipole" placed in the esophagus of living subjects have been determined. It is recognized that a dipole so located is not entirely comparable to the heart in relation to points in the frontal plane, especially as regards the lateral wall of the left ventricle and its relation to the left shoulder. This objection has been overcone in part by selecting some patients in whom the esophagus deviated to the right or left at about the level of the heart.

Materials and Method

Twelve patients with chronic disease or convalescent after an acute illness were selected for study. A variety of body types and nutritional states was represented.

Smooth round German silver disks, 5 mm. in diameter, were fixed on the wall of a stomach tube near its end and were connected to an input circuit by insulated copper wire inside the tube. In six experiments the electrodes were separated by a distance of 6 cm. and in the remainder of the experiments by 2 cm. In all experiments the tube was

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inserted a distance of 30 cm. or more as measured from the nose to the proximal electrode.

Essentially constant direct current of about 0.4 milliampere was supplied to the disk electrodes by a 45 volt battery through a circuit with slightly more than 100,000 ohms resistance. With this circuit, no effects attributable to polarization at the disk electrodes were apparent.

The position of the electrodes in the thorax was determined fluoroscopically and with spot films, and in some cases by frontal and lateral teleoroentgenograms. Observations on models with the electrodes in known positions indicated that the methods employed were accurate to within 5 degrees. The back electrode was placed in a position midway between the positive and negative electrodes of the esophageal dipole under fluoroscopic observation.

The leads recorded included the three standard leads and bipolar leads from the back and right arm, left arm and left leg, respectively. The standard leads were obtained on all subjects and bipolar leads from the back and right arm, left arm and left leg were obtained on 10 subjects.

While each lead was being recorded, the input circuit to the esophageal dipole was closed and opened at least nine times by means of a switch. Displacements of the baseline, similar to those which occur when an electrocardiogram is standardized, resulted. These were measured to the nearest 0.2 mm. and corrected for differences in standardization. The values so obtained were plotted on a triaxial reference system applied to each plane of the tetrahedron, to yield vector quantities which, if each apex of the tetrahedron is adequately remote from the dipole, should correspond in direction to the actual position of the dipole projected on the appropriate plane surface.

Results

Table 1 summarizes the data relative to the frontal plane. The actual position of the dipole projected onto the frontal plane as determined by radiographic means is expressed in degrees from the horizontal and is compared with the direction of vectors constructed from the deflections in the standard leads. As shown, these directions differ by 10 degrees or less.

In one experiment (No. 9) an almost horizontal input was obtained with the electrodes in contact with the fundus of the stomach. As was the case with more nearly vertical positions of the dipole, there was close agreement between actual dipole position and vectors plotted from the standard lead deflections.

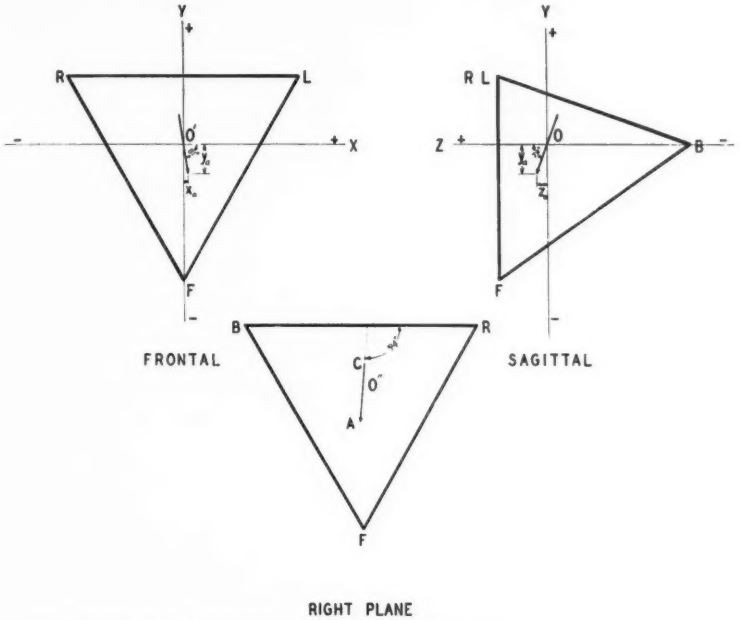

FIG. 1.—Projections of a dipole onto frontal, sagittal and right planes of the equilateral tetrahedron reference system.

Tables 2 and 3 summarize the data relating to the back electrode. From the frontal and sagittal positions of the dipole as determined radiographically, the projected position of the dipole in the right plane of the tetrahedron was calculated. A typical case is illustrated in figure 1. Projections of the dipole onto

TABLE 1.—*Direction of Vectors in Frontal Plane*

Experiment Number	Direction of Dipole Projection	Direction of Vectors Plotted from Leads			Maximal Difference
		I and II	I and III	II and III	
	degrees				
1	90	90	90	89	1
2	93	90	90	86	7
3	94	96	96	96	2
4	76	75	75	75	1
5	83	73	75	80	10
6	88	90	90	84	4
7	90	90	90	89	1
8	100	90	90	102	10
9	6	9	8	9	3
10	81	81	82	87	6
11	80	84	85	90	10

TABLE 2.—*Direction of Vectors in Right Plane*

Experiment Number	Calculated Direction of Dipole Projection	Direction of Vectors Plotted from Leads			Maximal Difference
		II and BF	BR an BF	BR and II	
	degrees				
1	116	109	99	97	19
2	80	84	90	90	10
3	107	93	107	115	14
4	118	125	120	115	7
5	139	136	137	141	3
6	73	75	75	76	3
10	94	98	85	86	9
11	81	80	73	75	8
12	118	117	107	105	13

the frontal and sagittal planes (determined radiographically) and onto the right plane (calculated) are shown. In this figure, the frontal plane triangle *RLF* is perpendicular to the *z* axis, positive coordinates are measured as indicated, and x_a, y_a and z_a are the coordinates of the lower end of the dipole. The center of the dipole, *O*, is at the intersection of the coordinate axes and at the center of the tetrahedron. *CA* is the projection of the dipole onto the right plane. Angle α between *CA* and edge *RB* in the right plane was calculated by means of the formula:

$$\alpha = \left\{ \arccos \frac{\frac{1}{\sqrt{3}}\left(-x_a + \frac{2}{\sqrt{3}}y_a + \frac{2}{\sqrt{6}}z_a\right)}{\sqrt{(x_a^2 + y_a^2 + z_a^2) - \frac{2}{3}\left(x_a + \frac{y_a}{\sqrt{3}} + \frac{z_a}{\sqrt{6}}\right)^2}} \right\} - 30°.$$

Positive angles were measured clockwise from the reference axis *RB*, as is customary in electrocardiography.

The directions of the dipole projections in the left plane were not calculated but have been

TABLE 3.—*Direction of Vectors in Left Plane*

Experiment Number	Calculated Direction of Dipole Projection	Direction of Vectors Plotted from Leads			Maximal Difference
		III and BF	BR and BF	BR and III	
	degrees				
1	116	109	107	106	10
2	82	87	90	90	8
3	115	99	104	107	16
4	110	114	117	119	9
5	130	134	131	121	9
6	74	73	62	69	12
10	89	101	90	90	12
11	76	80	72	74	4
12	110	105	112	117	7

estimated with the use of a tetrahedron model. It was not considered necessary to make these calculations, since in the case of the right plane the directions as estimated with a model were essentially the same as those calculated.

The direction of the dipole projection in these planes has been expressed in degrees from the horizontal and has been compared with the direction of vectors plotted from the deflections in bipolar leads from the apices of the appropriate plane. The greatest discrepancy between the actual dipole position and the constructed vector direction was nineteen, and the average discrepancy was ten degrees. Since it is likely that the greatest errors in determining the actual dipole position were made in the case of the sagittal angles, these discrepancies would seem to be in the range of those encountered in the frontal plane.

Discussion

Experience with spatial vectorcardiography to date offers encouragement for the eventual clinical usefulness of that method, but preceding more extensive use it is desirable to have information relative to the errors involved.

The results of this study indicate in a gross fashion that a point on the back may be considered electrically remote from a dipole in the region of the heart with approximately the same error as is involved in considering the apices of the Einthoven triangle remote. Since the Einthoven concept is unquestionably valuable in frontal plane electrocardiography, it seems likely that the tetrahedron will prove useful for spatial studies of the action current.

Recently Wilson and his associates[3] have reported the results of preliminary studies with the use of a dipole on the external surface of the thoracic wall to quantitate the error of the Einthoven hypothesis in individual cases and to investigate the possibility of constructing an Einthoven triangle for a given subject. It is hoped that further studies of a similar nature may yield more exact information regarding the errors inherent in applying reference systems to the human body than are now available and may suggest methods for reducing these errors.

Summary

1. The validity of the tetrahedron reference system has been investigated with the use of a "dipole" in the esophagus of living subjects to produce an electric field in that subject.

2. Results indicate in a gross manner that a point on the back can be considered electrically "remote" from the esophageal dipole with an error equivalent to that involved in considering the apices of the Einthoven triangle remote.

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An Approximate Derivation for Stereoscopic Vectorcardiograms with the Equilateral Tetrahedron

By J. A. Cronvich, M.S., J. A. Abildskov, M.D., C. E. Jackson, M.D., and G. E. Burch, M.D.

A simple electrode arrangement, which permits the derivation of stereoscopic projections of the spatial vectorcardiogram, eliminates the need for constructing models. The visual impression obtained from this method agrees satisfactorily with that from carefully constructed models.

IN THE study of spatial vectorcardiography in this laboratory it has been the practice to construct wire models of the spatial vectorcardiogram from its frontal and sagittal projections which are presented on two cathode-ray tubes.[1, 2] These three-dimensional models seemed necessary to facilitate visualization of the spatial characteristics of the vectorcardiogram, but their construction entailed considerable time and care. To avoid the necessity of constructing such models, we have recently developed a simple electrode arrangement which permits satisfactory stereoscopic views of the spatial vectorcardiogram to be photographed directly from two cathode-ray tubes.

One plane is rotated by approximately three degrees with respect to the frontal plane by displacement of its right apex backward. The other plane is rotated through an equal angle by displacement of its left apex backward.

In the arrangement devised each cathode-ray tube presents the projection of the vectorcardiogram onto a plane displaced slightly from the frontal plane. Since the electrical derivation of these planes involves the application of only four electrodes, right arm (RA), left arm (LA), left leg (F), and back (B), which form the apices of the equilateral tetrahedron, this method is simpler than that of using adjacent electrodes employed by Schellong[3] and modified by Vastesaeger and Rochet.[4] The shift of these planes from the frontal plane is obtained by means of the resistors connecting RA, LA and B in the superior plane (see fig. 1). RA′, LA′ and F are the apices of the plane whose right apex is shifted backward along RA-B, and RA″, LA″ and F are the apices of the plane whose left apex is shifted backward along LA-B. The frontal plane projection of the vectorcardiogram is obtained by using standard Lead I connections to produce horizontal deflection on the oscilloscope and the unipolar limb potential from the foot (VF) to produce vertical deflection.[1, 2] Similarly, stereoscopic views are obtained by using the potential difference between RA′ and LA′ in the plane RA′-LA′-F to produce horizontal deflection on the oscilloscope and VF to produce vertical deflection. The recorded pattern from this oscilloscope is viewed with the left eye. In the plane RA″-LA″-F the potential difference between RA″ and LA″ is used to produce horizontal deflection and VF to produce vertical deflection. The recorded pattern from this oscilloscope is viewed with the right eye.

Ideally, vertical deflection in both RA′-LA′-F and RA″-LA″-F should be obtained from central terminal to foot derivations in each of these planes. However, the error introduced by our approximate derivation is probably small compared with errors inherent in the use of such an idealized reference frame as the tetrahedron. Many refinements are obvious but the stereoscopically obtained spatial vectorcar-

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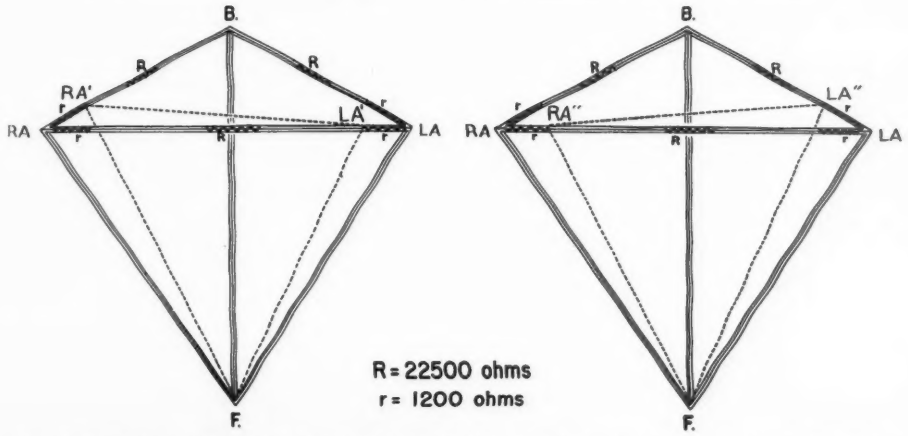

FIG. 1 Equilateral tetrahedra showing derived planes used in stereoscopic vectorcardiography.

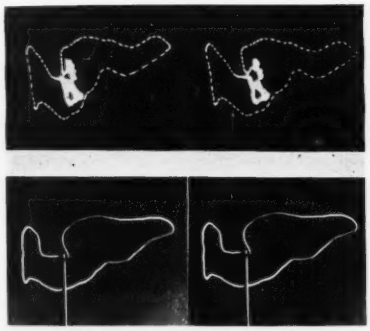

FIG. 2A. (*Above*) Stereoscopic vectorcardiogram recorded by the method presented in this paper. *B*. (*Below*) Stereoscopic photograph of wire model constructed from frontal and sagittal projections of the vectorcardiogram shown in *A*.

diograms appear to be similar to the wire models carefully constructed from the frontal and sagittal projections. Possible modifications of this method include elimination of the 1200-ohm resistors in the frontal plane so that RA″ coincides with RA and LA′ coincides with LA, use of separate central terminal deviations in each plane for vertical deflection, and variation of the angles between the planes by changing the resistors used in the derivations. It may be possible to obtain the last of these in a more flexible manner if the electrodes are suitably located on the surface of a homogeneous sphere of resistive material.

A representative stereoscopic vectorcardiogram obtained by this method and stereoscopic photographs of a wire model constructed from the frontal and sagittal projections of the same vectorcardiogram are shown in figure 2.

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Comparison of Heart Vectors Calculated with Different Systems of Leads

By RICHARD MCFEE, M. S.

Einthoven's equations for the heart vector are based on the assumption that voltages measured across the trunk horizontally (or vertically) are proportional to the horizontal (or vertical) component of the dipole moment of the heart's electromotive forces. One necessary consequence of this assumption is that all the horizontal (or vertical) voltages be similar, and that heart vectors calculated with different sets of these voltages and appropriate formulas be the same. An experimental test of this predicted result is described in this paper.

INTRODUCTION

THE EXPERIMENTAL data, formulas and procedures discussed in this paper represent an effort to measure the *errors* involved in the use of the equations based on the Einthoven triangle. It was hoped that by comparing vectors calculated from several different systems of leads taken on normal human subjects a reliable estimate of the errors involved in the application of these equations and a rational basis for judging arguments about their validity would be obtained. The investigation of the natural field of the heart eliminates many objections applicable to experiments involving artificial electromotive forces, models and mathematical approximations. The original idea was that the errors resulting from the use of the Einthoven formulas would be of about the same magnitude as the differences between the heart vectors calculated from the different systems of leads, but it was eventually realized that this view was not sound, for there could be perfect agreement among the vectors of the different systems of leads even with pronounced variations in the conductivity of the tissues in and around the heart. Moreover, there would be no differences between those vectors if their vertical and horizontal components found with the different systems of leads were equally in error. For these reasons it was not possible to equate the differences among the vectors to the errors of the formulas, although it seems probable that these differences are a rough index of the accuracy of the latter.

The comparison of these heart vectors is discussed in a later section of this article. The appendix gives equations for calculating the errors in the vectors resulting from inaccurate measuring of the lead voltages. These equations were developed for the purpose of estimating to what extent the differences between corresponding vectors are due to errors in measurement and to what extent they are due to errors inherent in the method of calculating these vectors. They can be applied to any calculation of the heart vector based on Einthoven's triangle and have been found useful in finding the errors in measurements of the ventricular gradient.

METHOD

Three different systems of leads were used. The first was the usual Einthoven system. The second consisted of an inverted Einthoven triangle formed by two electrodes on the left and right sides of the waist halfway between the ribs and the pelvic bone, and a third electrode on the right side of the neck. The third system included a horizontal lead taken from electrodes on opposite sides of the chest about two-and-one-half inches below the armpits, and a vertical lead taken from between the neck electrode to an electrode on the abdomen about an inch below the navel. These lead systems are shown in figure 1.

All the voltages of each lead system were recorded simultaneously on one film, using a three-channel

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vacuum tube electrocardiograph. The film speed was 150 mm. per second, and the sensitivity was about 2 cm. per millivolt. A transparent and finely divided precision ruler was used to measure the vertical distance of the trace of the QRS deflections from points on the baseline separated by intervals of five milliseconds. The time measured voltages were found by multiplying each measurement by the calibration factor for the corresponding channel. These voltages were "adjusted" to their most probable values by using the formulas given in the appendix.

The usual Einthoven equations,

$$\alpha = \text{arc tan}\frac{2e_2 - e_1}{e_1\sqrt{3}} \qquad M = \frac{e_1}{\cos\alpha} \qquad (1)$$

were used for the two triangle systems, and the equations

$$\alpha = \text{arc tan}\frac{e_v}{\frac{1}{2}e_H} \qquad M = \sqrt{(e_v)^2 + (\tfrac{1}{2}e_H)^2} \qquad (2)$$

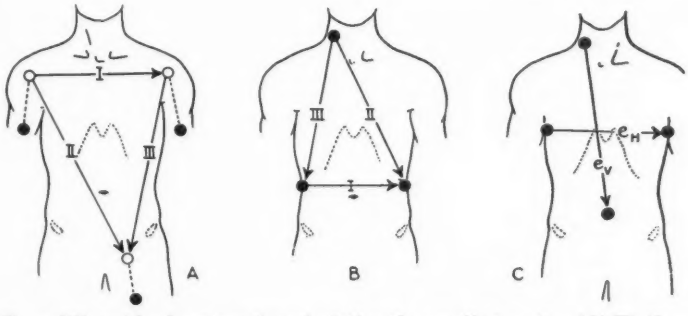

FIG. 1. Three different lead systems for calculating the manifest vector: (*A*) Einthoven triangle; (*B*) inverted Einthoven triangle; (*C*) vertical-horizontal lead system.

were used for the vertical-horizontal lead system. The horizontal voltage was arbitrarily divided by two, because the chest electrodes were nearer the heart than the arm and waist electrodes, for it was noted that the voltage across the chest was generally about twice as large as the corresponding voltages of the triangle systems; that is, of Lead I and the waist lead. The probable errors of the computed direction and the manifest magnitude of the cardiac vector due to mistakes in measurement were calculated by using the formulas given in the appendix. The time in the cardiac cycle of each measured voltage, with respect to an instant slightly before the onset of the complex, was determined. It is possible that errors as great as three or four milliseconds, one way or the other, were made in doing this.

RESULTS

Graphs showing the data collected in this way are reproduced in figure 2. These superimposed curves give the manifest magnitude of the heart vector and the angle which defines the direction for each subject plotted against time. It will be noted that on each graph two curves are drawn for the ordinary Einthoven system of leads. These two curves were obtained from different heart beats and show the variations that result from using different beats for the calculations under consideration. It can be seen from the graphs that in each case these variations are relatively small.

These graphs show in detail the information obtained from the experiments. Of particular interest are the differences in angle and in magnitude between the highest and lowest curves of the graphs. As was mentioned in the introduction, these differences may be thought of as a rough index of the accuracy of the Einthoven formulas. For the convenience of the reader, average and maximum values for the differences of angle, along with average values for their probable errors and variations between beats are given in table 1. Corresponding differences in the magnitude of the vectors were not tabulated, because it was felt that the small errors made in locating the time scales would make the resulting differences misleadingly large.

In obtaining these numbers, attention was restricted to that portion of the QRS complex where the manifest magnitude exceeds one quarter its maximum value. This was done to minimize the large errors in measurement which are always associated with small deflections. The differences between the highest

and lowest curves were determined at instants five milliseconds apart in this interval, and the maximum and average of the results were tabulated. The differences between the two curves calculated with the Einthoven lead system were determined at the same instants, and also averaged and tabulated. The probable error figure was obtained by averaging the calculable probable error of all measurements in the interval considered.

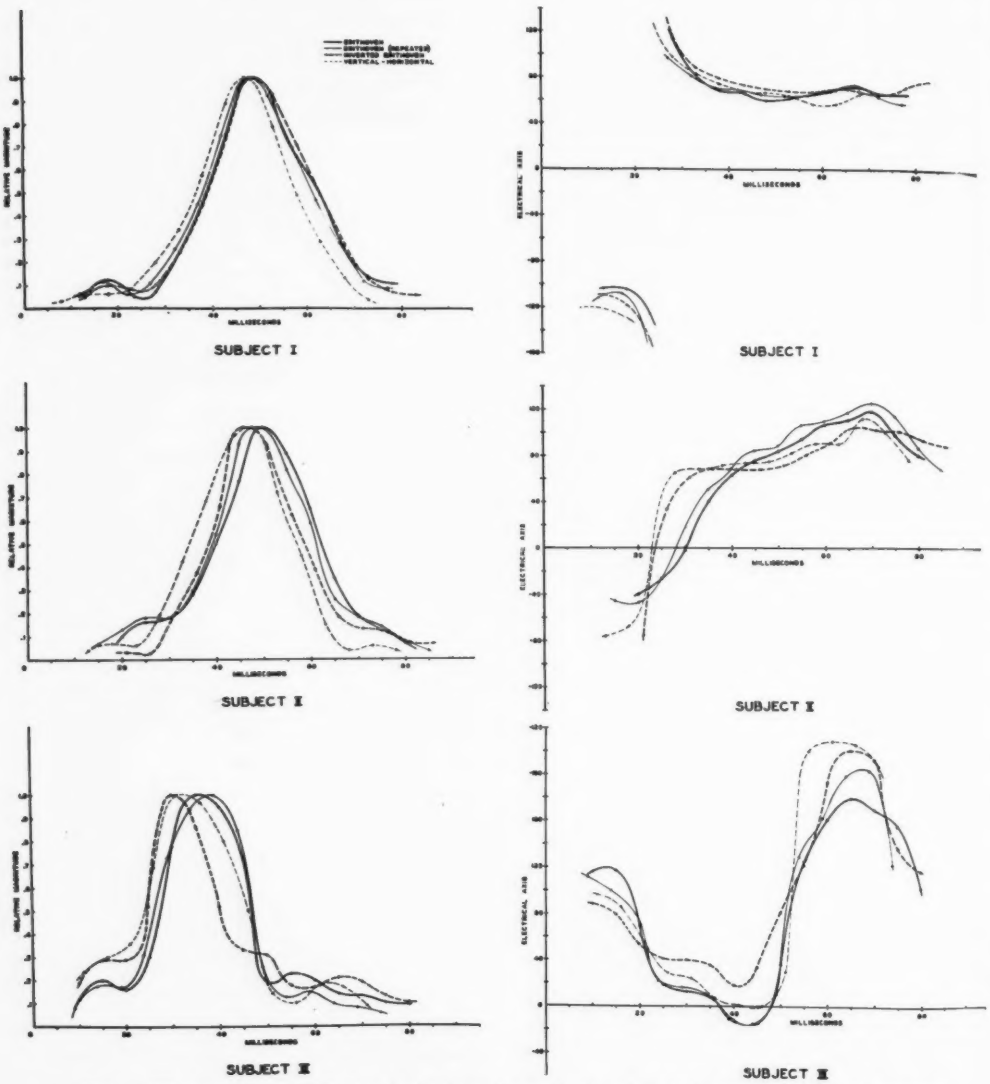

FIG. 2. Time graphs of QRS electrical axis and magnitude calculated from different lead systems for three subjects. To indicate variations from beat to beat, calculations were made with the Einthoven triangle lead system for two different complexes.

DISCUSSION

There is no doubt that the inverted Einthoven triangle and the vertical-horizontal lead systems used here are completely arbitrary, as

are the equations which accompany them. But the Einthoven triangle itself was chosen on an arbitrary basis, and there seems to be no basic reason why any one of the lead systems should be considered superior to any of the others. The equations for the leads may be chosen

TABLE 1.—*Tabulated Values for the Differences Between the Curves in Figure 2.**

	Maximum Difference Between Highest and Lowest Curve	Average Difference Between Highest and Lowest Curve	Average Probable Error	Average Difference Between Curves of Same Lead System withDifferent Beats
Subject 1	14.9°	9.9°	1.0°	2.4°
Subject 2	69.8°	25.8°	1.9°	9.0°
Subject 3	58.8°	30.8°	1.9°	5.4°

* For detailed explanation, see text.

more rationally using the concept of a lead vector recently advanced by Burger and Van Milaan[1] and finding the components of this vector with a suitable model, as they did. Or they may be chosen using a human subject and artificially inducing a dipole, as was done by Wilson and his associates.[2] But in any case, by comparing heart vectors calculated from different systems of leads with formulas obtained by such methods, we can test their validity when applied to human subjects, and in this manner find out which ways of calculating the heart vector are best.

The procedure of comparing vectors calculated with different lead systems suggests a method of constructing a lead which is more uniformly sensitive to electromotive forces generated in different parts of the heart. This is done by using two or more electrodes for each end of the lead and averaging their potentials. The electrodes are located so that the electromotive forces they emphasize are in opposite portions of the heart. This emphasis, as a result, will be cancelled out when their potentials are averaged. Actual averaging networks are constructed by connecting the electrodes with equal resistances to the same point, as is done with the central terminal. It is easily

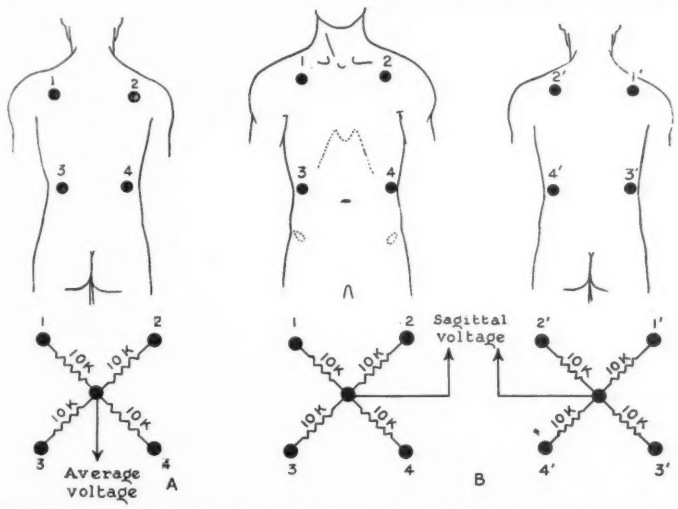

FIG. 3. Two electrical averaging systems. (*A*) averaging network for Wilson's tetrahedron. (*B*) Cubical electrode system, showing averaging network for obtaining the sagittal component. Identical networks are used to find the other two components.

shown that the potential of this point will be the average. The requirement that the sum of the currents flowing into the point be zero (Kirchhoff's current law) is equivalent to the equation

$$\frac{V_1 - V_a}{R} + \frac{V_2 - V_a}{R} + \cdots + \frac{V_n - V_a}{R} = 0 \qquad (3)$$

$$n = 2, 3, 4, \cdots$$

where V_1, V_2, . . . V_n are the potentials of the n electrodes and V_a is the potential of the output terminal. Solving this equation for V_a gives

$$V_a = \frac{1}{N}(V_1 + V_2 + \cdots + V_n) \qquad (4)$$

which is the desired average.

Some examples of these averaging systems are shown in figure 3. Part *A* of this figure shows an application to the tetrahedron of Wilson, Johnston and Kossmann.[3] The central point of the averaging network replaces the usual sagittal electrode of the tetrahedron. In this way a sagittal electrode is obtained which is effectively more "remote" from the heart. Part *B* shows a symmetrical system for obtaining all three components of the heart vector with eight electrodes. Each component is the average of four bipolar leads. If the output voltages of this latter system are considered directly proportional to the three components of the heart vector, proper weighting factors for each component may be introduced by adjusting the gain of the amplifiers. If Burger's equations for the output leads are used, simple resistive networks may be constructed for obtaining linear combinations of the output voltages, and they can be adjusted so that the combined voltages are proportional to the desired components.

Summary

The instantaneous magnitude and direction of the cardiac vector is calculated for the QRS complex of three subjects using the Einthoven lead system, an inverted Einthoven lead system and a two lead "vertical-horizontal" lead system. Comparison of the time graphs of the results gives a qualitative idea of the accuracy of the Einthoven formulas.

Equations are given for estimating the probable errors in the manifest vector resulting from inaccurate measurement of the voltages of the leads.

Appendix

Equations for estimating the errors resulting from inaccurate measurement of the voltages of the leads:

These formulas may be used only for calculations based on the ordinary Einthoven triangle.

$$\text{Probable error in angle } (\alpha) = \pm 40 \frac{\Delta}{M} \text{ degrees} \qquad (1)$$

where

$$\Delta = I + III - II \qquad (2)$$

$$\text{Probable error in manifest magnitude } (M) = \pm 80 \frac{\Delta}{M} \text{ per cent} \qquad (3)$$

The probable error of a measurement is that quantity which will, on the average, exceed and be exceeded by half of the actual errors.

The influence of measurement errors may be reduced about forty percent by "adjusting" the lead voltages so that they conform with Einthoven's Law. The equations for the adjusted values are

$$\begin{aligned} I_a &= I - \tfrac{1}{3}\Delta \\ II_a &= II + \tfrac{1}{3}\Delta \\ III_a &= III - \tfrac{1}{3}\Delta \end{aligned} \qquad (4)$$

When these adjusted lead voltages are used the formulas for the measurement of errors become

$$\text{Probable error in } \alpha = \pm 25 \frac{\Delta}{M} \text{ degrees} \qquad (5)$$

$$\text{Probable error in } M = \pm 50 \frac{\Delta}{M} \text{ per cent} \qquad (6)$$

The equations for the probable error of the measurements are likely to be in error by as much as 100 per cent, but they are constructed so that on the average half the results obtained with them will be larger than the true probable errors, and half will be smaller.

The derivations of these formulas are fairly lengthy and will not be given here. The general idea is as follows. It is assumed that there is an equal distribution of random errors in the measured values of all lead voltages. The probable error of the Δ distribution, ϵ_Δ, can then be shown to be related to the probable errors in the individual leads, ϵ_L, by

$$\epsilon_\Delta = \sqrt{3}\,\epsilon_L \qquad (7)$$

The probable errors in the individual leads are found by letting

$$\epsilon_\Delta = \Delta \qquad (8)$$

This assumption (8) leads to results which will be too large half the time and too small the other half. The probable errors for α and β are then found using Einthoven's equations and the conventional formula for the propagation of errors.

$$\text{Probable error of } F(x, y, z) = \sqrt{\left(\frac{\partial F}{\partial x}\epsilon_x\right)^2 + \left(\frac{\partial F}{\partial y}\epsilon_y\right)^2 + \left(\frac{\partial F}{\partial z}\epsilon_z\right)^2} \qquad (9)$$

where ϵ_x, ϵ_y and ϵ_z are the probable errors of x, y, and z.

When adjusted values for the lead voltages are used, errors of each lead must be identified and added linearly, instead of in a root mean square fashion as is done for errors from different leads.

The adjusted values for the lead voltages given in equation (4) are such that they simultaneously satisfy Einthoven's law and the requirement that the root mean square of the difference between them and the measured values ("residuals") be minimum. It can be shown that these are the most probable values of the lead voltages.

ACKNOWLEDGMENT

The author wishes to express his gratitude to Dr. Frank N. Wilson for his helpful suggestions in the preparation of this paper.

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Chronic Pericarditis with Effusion

By PAUL S. BARKER, M. D., AND FRANKLIN D. JOHNSTON, M. D.

Pericarditis with effusion is sometimes a chronic disease, an exception to the general rule that it is acute rather than chronic. Illustrative cases are described. Features of interest are the prominent third heart sound, the large P waves, the thrombi in the right atrium, the progression to constrictive pericarditis, and the resemblance in several respects to mitral stenosis.

PERICARDITIS is commonly classified as acute or chronic. Pericarditis with effusion is regarded as a form of acute pericarditis and not as a form of chronic pericarditis.[1, 2] This is undoubtedly correct in nearly all instances. There are, however, occasional exceptions in which pericarditis with effusion follows a chronic course of many months, notable for the complete absence of the features of acute disease. The purpose of this report is to call attention to this less common type of chronic pericarditis with effusion, to describe three illustrative cases and to comment on several features of special interest.

CASE REPORTS

Case 1.—R.Z. (U.H. 576097) was an 18 year old student when first seen on September 4, 1945. He had enjoyed good health until the age of 13 years when he had pneumonia and was told that he had rheumatic heart disease. His activities were restricted upon the advice of his physician, but he soon developed fatigue and shortness of breath upon moderate exertion which gradually increased in severity. There was no history of cardiac pain, palpitation or edema, or of previous rheumatic fever, growing pains, chorea or cyanosis, although he had frequent sore throat during childhood.

Physical examination revealed a healthy appearing youth with gross cardiac enlargement, dulness extending 6 cm. to the right and 13 cm. to the left of the mid-sternal line. The pulmonary second sound was slightly accentuated, but there was no palpable impulse, heave, shock or thrill, and no friction, murmur or gallop. The rhythm was normal. The neck veins were slightly distended. The lungs were clear. The liver was enlarged to 7 cm. below the right rib margin, but there was no ascites. Minimal edema of the ankles was present. There were no signs of thyroid disease. There was a paradoxical pulse; the blood pressure was 96–85/6C.

The hemoglobin was 17.9 Gm., the blood Kahn test was negative. The electrocardiogram showed broad, notched, deformed auricular deflections (fig. 1). X-ray examination revealed moderate enlargement of the heart with a globular contour, and minimal left hydrothorax. A review of nine previous films, beginning on January 7, 1941, showed progressive increase in size of the heart, except in the films of September 18, 1943 and September 4, 1945 which showed decrease in cardiac size.

Upon limited activities, digitalis and salt restriction the patient's course was satisfactory until the summer of 1947, when dyspnea and edema increased and ascites developed. Auricular fibrillation appeared and the course was progressively downward. On November 19, 1947 the findings were characteristic of constrictive pericarditis. There was only slight enlargement of the heart; it was definitely smaller than in 1945. In spite of the great risk, pericardiectomy seemed to be the only hope. The patient died on December 12, 1947, sixteen hours after operation. The temperature was normal throughout and the white blood cell count was normal.

The autopsy showed chronic constrictive pericarditis with partial pericardiectomy. There was polyserositis with active chronic fibrous pericarditis, pleuritis and peritonitis and "pericarditic pseudocirrhosis" of the liver. There was extreme atrophy of the muscle fibers of the ventricles, and hypertrophy of the atrial muscle. The mitral leaflets showed slight fibrous thickening, but no active inflammation and no vascularization and no stenosis. There was no evidence of rheumatic fever or tuberculosis.

Comment.

This patient showed clinical and roentgenographic evidence of pericarditis with effusion from 1941 through 1945, which by 1947 had progressed to constrictive pericarditis. There was no clinical evidence of active or acute disease during this time. The etiology of the pericarditis and polyserositis was not apparent; the pneumonia in 1940 might have been the cause. A feature of interest was the finding at autopsy of atrophy of the muscle fibers of the ventricles whose movements must have been greatly restricted by the diseased pericardium, and of hypertrophy of the auricular muscle.

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The broad, notched auricular deflections of the electrocardiogram made two years before death were of the type observed in mitral stenosis and in other conditions associated with auricular hypertrophy.

Case 2.—H.M. (U.H. 603646) was a 38 year old farmer, first seen on November 12, 1946, when he complained of swelling of the lower extremities. He stated that in 1943 he was kicked in the chest by a horse, following which he had dyspnea upon slight exertion and swelling of the feet, and that he was

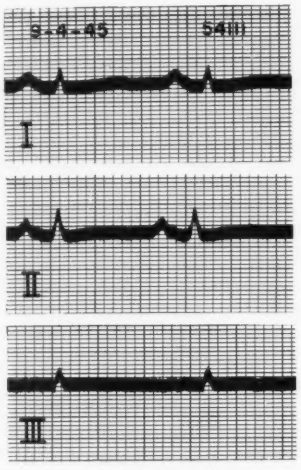


FIG. 1.—Case 1. Electrocardiogram of Sept. 4, 1945. Leads I, II and III. The P waves are broad and notched. The QRS deflections are of relatively small amplitude.

told that his heart was enlarged. He improved slowly until he was able to drive a truck, but he gave up farming. In December, 1944, he had several hemoptyses, followed by dyspnea upon slight exertion, fullness in the epigastrium and swelling of the ankles. From that time on he had recurring episodes of dyspnea and swelling of the feet and legs, improving under treatment by his physician but soon returning and becoming much more pronounced in the summer of 1946. There was no history of thoracic pain or palpitation, or of rheumatic fever, renal disease, scarlet fever or venereal disease.

Physical examination showed an obese man with massive edema of the legs, thighs and genitalia, but able to lie flat. The cardiac dulness was greatly enlarged, the sounds were distant but regular, a third sound in diastole was heard near the apex, but no friction or murmur was detected. The blood pressure was 105/80, the pulse was not paradoxical. The neck veins were distended, there were râles at the lung bases, and the liver was enlarged, but ascites was not present.

X-ray examination showed marked cardiac enlargement, pulsations were present but diminished, and the shape of the cardiac silhouette changed with change in position of the patient. The electrocardiogram showed large, broad P waves. The total serum proteins were 6.9 per cent, albumin 4.1 per cent and globulin 2.8 per cent.

The patient improved upon rest, digitalis and a diuretic regimen. It was thought at first that he

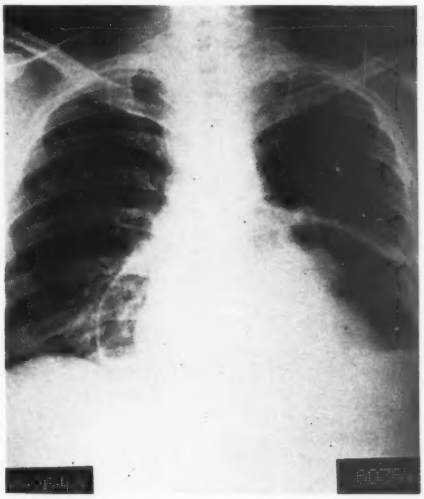


FIG. 2.—Case 2. X-ray of chest made on Aug. 26, 1948, immediately following the removal of 1650 cc. of fluid from the pericardial cavity and the replacement of 800 cc. of air. The thickened and distended parietal pericardium is widely separated from the heart by a moderate amount of residual fluid and the injected air. The cardiac shadow is somewhat enlarged.

might have rheumatic heart disease with mitral stenosis, but it later became apparent that he had pericarditis with effusion.

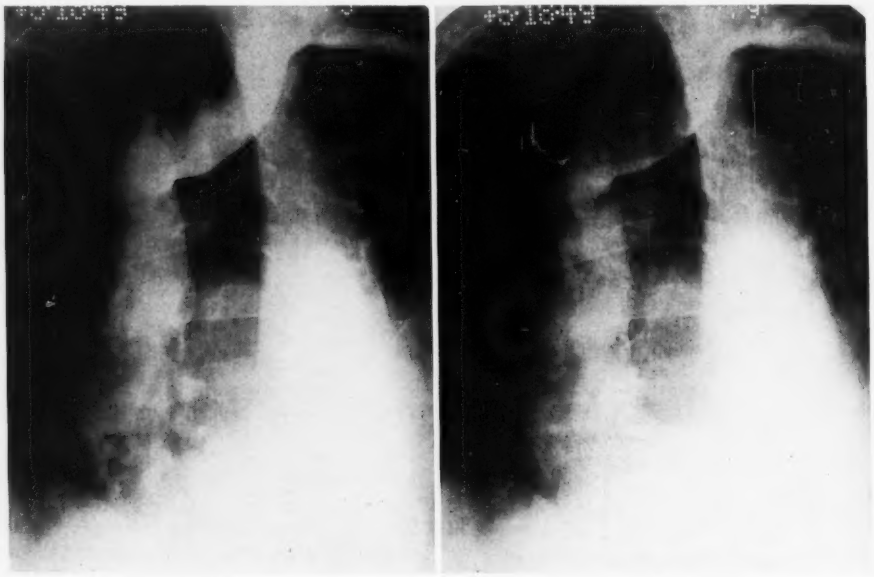
The patient got along fairly well during 1947 on digitalis, ammonium chloride intermittently, restriction of sodium intake and an occasional mercury diuretic, and although slight to moderate edema persisted he was able to work as a taxi cab driver. Early in 1948, however, auricular fibrillation appeared and dyspnea and edema became pronounced. In June, 1948 the edema was massive, orthopnea was present and there were râles at the lung bases. The cardiac findings were as before, with greatly enlarged cardiac dulness, auricular fibrillation, a third sound in diastole, and no murmur or friction. The serum albumin had declined to 3.0 per cent. In July and August, 1948, pericardial paracentesis was performed on five occasions; the needle encountered thickened pericardium (fig. 2). The amounts of fluid withdrawn varied from 325 cc. to 1650 cc.; it was clear and

straw colored at first, but later it was somewhat bloody. The fluid was negative for acid fast bacilli upon smears of concentrated preparations and cultures for pyogenic organisms were sterile.

Partial pericardiectomy was performed by Dr. John Alexander on August 27, 1948. Thickened, inflamed, parietal pericardium was removed from the anterior and left lateral aspects of the heart. All chambers of the heart appeared somewhat enlarged and especially the right atrium. The tissue removed showed evidence of acute fibrinous exacerbation of chronic fibrous pericarditis.

Improvement was transient. Edema soon became more pronounced and paradoxical pulse and ascites appeared. Auricular fibrillation, cardiac enlargement, and the diastolic third sound persisted. On December 8, 1949, Dr. Cameron Haight removed thickened epicardium from the left ventricle and much of the right ventricle, after which the amplitude of ventricular contractions was good. The right atrium was distended, motionless and quite firm; it was thought to contain an organized thrombus. The left auricle seemed normal. The tissue removed showed evidence of chronic fibrous pericarditis. There was only slight improvement following the second operation.

Throughout the period of observation from November, 1946 to December, 1948 the clinical course was that of a chronic disease. The temperature was normal on all occasions except transient elevations following the operations. The white blood cell count was 13,800 on November 15, 1946, but was normal a few days later and upon all subsequent determinations. The acute fibrinous change observed in the tissue removed at the first operation is attributed to the repeated tappings which preceded the operation.



A *B*

FIG. 3.—Case 2. Angiocardiograms made on May 18, 1949. (*A*) Film exposed 3.75 seconds after the appearance of the opaque media in the superior vena cava. This vessel is dilated and well filled. The right atrium is incompletely filled and presents a mottled appearance. Normally at this time the superior vena cava should be almost free of the opaque substance and the right atrium and ventricle should be well filled with it. (*B*) Film exposed 6.75 seconds after the appearance of the opaque media in the superior vena cava. The superior vena cava still appears dilated and well filled, while the right atrium is incompletely filled and mottled and the right ventricle is somewhat more completely filled with the opaque substance. Normally at this time the superior vena cava and right atrium should be completely free, and the right ventricle almost completely free of the opaque media. The examination is interpreted as showing dilatation and prolonged filling of the superior vena cava and slow and incomplete filling of the right atrium and right ventricle, compatible with the presence of a thrombotic mass in the right atrium. (Courtesy Dr. Melvin M. Figley)

Comment.

This patient was known to have had pericarditis with effusion continuously from No-

vember, 1946, to August, 1948, and it probably began in June of 1943. The cause of the pericarditis was not determined, but it might have been caused by the injury to the chest sustained in June, 1943. The clinical course was chronic leading finally to constrictive pericarditis. The failure to improve satisfactorily after partial pericardiectomy on two occasions may be attributed in part to the presence of a mass, probably a thrombus, in the right atrium. It is of interest that the angiocardiogram revealed dilatation and prolonged filling of the superior vena cava and slow, irregular filling of the right atrium and right ventricle, suggesting a mass in the right atrium (fig. 3).

Case 3.--N.H. (U.H. 666733), a 33 year old taxi cab driver, was first seen on August 29, 1949, complaining of swelling of the abdomen. He stated that he was not accepted for military service in 1940 because of a "leaky heart." There had been no symptoms referrable to the heart until May, 1949. Then he developed shortness of breath upon exertion which was followed in two weeks by swelling of the abdomen and ankles. The dyspnea became more pronounced and orthopnea developed. There was no pain. The patient was hospitalized elsewhere, digitalis was given and abdominal paracentesis was performed on three occasions, but improvement was only transient. There was no history of rheumatic fever, growing pains, chorea or venereal disease.

Physical examination showed a well developed man with pronounced enlargement of the area of cardiac dulness, auricular fibrillation, a systolic murmur over the entire precordium, and a diastolic third sound near the apex. There was no friction rub. The blood pressure was 125/70. The neck veins were distended. The lungs were clear. The liver was enlarged and ascites was present. There was no edema of the legs.

The electrocardiogram showed auricular fibrillation and deflections of small amplitude. X-ray examination showed marked, generalized, cardiac enlargement with somewhat diminished pulsation of the left border. The blood Kahn test was negative. The blood count was normal. The urine was normal. The total serum proteins were 7.7 per cent, albumin 4.8 per cent and globulin 2.9 per cent.

It was thought at first that the patient might have rheumatic heart disease with mitral stenosis, but it soon became apparent that he had pericarditis with effusion. He improved somewhat upon rest, digitalis, ammonium chloride intermittently, and restriction of sodium intake, but the cardiac enlargement, the diastolic third sound, the distension of the neck veins, and enlargement of the liver persisted and the ascites soon returned. The pulse was paradoxical at times. On September 30 about 600 cc. of clear, straw-colored fluid were removed by pericardial paracentesis, after which the patient felt better without showing objective improvement. On October 31 the pericardium was tapped again and 400 cc. of fluid were removed. On November 8 a third tap yielded 320 cc. The fluid was clear and straw colored with a specific gravity of 1.017, and the spun sediment showed 10 to 12 cells per high power field, mostly lymphocytes; no acid fast bacilli were found in smears of the concentrated preparations and cultures for pyogenic organisms were sterile.

On December 7, 1949, Dr. Herbert E. Sloan, Jr., performed a partial pericardiectomy. The distended pericardium was incised and several hundred cc. of fluid were aspirated. The heart appeared greatly enlarged, chiefly the right ventricle and right atrium. The right atrium was a bulging mass 10 by 5 by 4 cm., motionless and firm, thought to contain a thrombus. The left ventricle and left atrium seemed normal. The epicardium was smooth and glistening. The parietal pericardium was only slightly thicker than normal; it was widely resected. The tissue showed evidence of chronic fibrous pericarditis.

The patient was somewhat improved following operation, but his symptoms were not entirely relieved. The cardiac enlargement, auricular fibrillation, systolic murmur and diastolic third sound persisted. The venous pressure was lower, but soon rose again and the ascites returned. The temperature was normal throughout except during the four days following operation, and the white blood cell count was normal repeatedly.

Comment.

This patient had pericarditis with effusion from May to December, 1949. The disease ran a chronic course without pain, fever or leukocytosis, but had not progressed to the stage of constrictive pericarditis by the time of operation in December. The etiology of the pericarditis was not apparent.

Discussion

These three cases are clearly examples of pericarditis with effusion in which the disease was chronic. The painless course of seven months to four years or longer, without fever or leukocytosis, and the histological evidence of chronic fibrous pericarditis, point to chronic disease. They cannot properly be classified as acute pericarditis. Although they are exceptions to the general rule that pericarditis with effusion is acute rather than chronic, it is thought that cases of this type are not rare. They present several features of interest.

In none of these cases was the etiology of the pericarditis apparent. The clinical and histological findings and study of the fluid yielded no evidence of tuberculous or rheumatic etiology. In the first patient the disease followed pneumonia the type of which is not known, while in the second case the symptoms followed an injury to the chest. In the third instance there was no preceding illness. The fluid resembled a transudate, but the tissue removed showed evidence of inflammatory disease. All three patients were relatively young, white males.

The symptoms were dyspnea, edema and swelling of the abdomen, compatible with cardiac tamponade but not differing greatly from those commonly encountered in mitral stenosis with congestive failure. The physical signs were those of pericardial effusion with cardiac tamponade, although at times paradoxical pulse was not detected.

A feature of special interest was the prominent third sound in early diastole in two of the patients, best heard near the apex. Attention has been called to a similar sound occurring in chronic constrictive pericarditis, and to its superficial resemblance to the murmur of mitral stenosis,[3] but its occurrence in pericarditis with effusion has not been emphasized.

The electrocardiograms obtained in two of the patients during normal rhythm, before the onset of auricular fibrillation, showed large, broad or notched auricular deflections like those commonly observed in mitral stenosis. All eventually developed auricular fibrillation. The x-ray examination revealed the cardiac enlargement, but was of little help in establishing the diagnosis of pericardial effusion. It could be decisive if angiocardiography were employed, and in distinguishing pericardial from pleural effusion if films were made after the withdrawal of fluid and its replacement by air.

The diagnosis of pericarditis with effusion was not at first apparent in any of these cases. The two patients with diastolic third heart sounds were thought at first to have mitral stenosis. The absence of the other signs commonly present in mitral stenosis, such as an accentuated apical first sound or pulmonic second sound, and a more careful evaluation of all of the findings soon led to the correct diagnosis. The tendency for auricular fibrillation to occur, and for the disease to progress to chronic constrictive pericarditis is perhaps to be expected.

The finding in two of the patients at operation of evidence of a large, organized thrombus in the right atrium was of interest. It is probable that the mass partly filling the right atrium offered an impediment to the return of blood to the right heart and thus contributed materially to the elevation of the venous pressure and to the formation of ascites and edema. Indeed, in the second case, the angiocardiograms supported this view, showing dilatation and prolonged filling of the superior vena cava and slow, irregular filling of the right atrium and right ventricle. This may explain this patient's failure to improve satisfactorily after wide resection of thickened pericardium on two occasions sufficient to permit good ventricular contractions. It is thought that the thrombus formation in the atrium was secondary to the pericardial disease and the auricular fibrillation, and not primary.

Summary

1. Pericarditis with effusion is sometimes a chronic disease. Although this is an exception to the general rule that pericarditis with effusion is acute rather than chronic, such cases are probably not rare. Three illustrative cases of unknown etiology are described.

2. Features of special interest are the occurrence of a prominent third heart sound in early diastole near the apex, the observation of large, broad or notched P-waves in the electrocardiograms, the presence of large, organized thrombi in the right atrium, and the tendency to progress to chronic constrictive pericarditis.

3. The diagnosis is often not at first apparent, the condition being most readily confused with mitral stenosis.

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ABSTRACTS

BACTERIAL ENDOCARDITIS

Moncke, Co.: Subacute Bacterial Endocarditis with Unusual Course. Deutsche med. Wchnschr. **74:** 1425 (Nov.) 1949.

The author observed 20 atypical cases of subacute bacterial endocarditis. The unusual features were repeatedly sterile blood cultures, normal or only subfebrile temperature, a lack of microembolism and, in more than half of the cases, signs of a diffuse glomerulonephritis. In 12 cases the diagnosis of subacute bacterial endocarditis was confirmed at autopsy; however, cultures from the valves remained sterile. Because the clinical impression was rather that of a focal infection than that of endocarditis lenta, the author suggests the name type F for this group, which he feels will be observed whenever a large number of cases of subacute bacterial endocarditis are studied.

PICK

BLOOD COAGULATION

Rosenthal, R. L.: Blood Coagulation in Leukemia and Polycythemia; Value of the Heparin Clotting Time and Clot Retraction Rate. J. Lab. & Clin. Med. **34:** 1321 (Oct.), 1949.

One-half of patients with chronic myelogenous or lymphatic leukemia showed a prolongation of the heparin clotting time and a decreased clot retraction rate and a slight thrombocytopenia. The severity of these defects was closely related to the degree of hemorrhagic symptoms. Leukemic patients without this clotting defect and with normal values for the coagulation tests showed no evidence of bleeding.

Of the untreated patients with polycythemia vera and elevated hematocrits, 33 per cent showed increased clot retraction rates and platelet counts elevated above 400,000. A similar percentage of these patients had histories of either thrombosis or hemorrhage. These studies indicate that the elevation in the platelet count is the most important single factor in the occurrence of both hemorrhage and thrombosis in polycythemia. Therapy should be directed toward the rapid reduction of an increased number of platelets.

The heparin clotting time appears to be the most valuable single test or index of coagulation. It is readily adaptable to both routine clinical and experimental use and it enables the detection of either abnormally increased or decreased clotting ability.

MINTZ

Battle, W. D., Capps, R. T., Orth, O. S., and Meyer, O. D.: The Effects of 4-Hydroxycoumarin Anticoagulant No. 63 upon the Prothrombin Time in Dogs and Human Beings. J. Lab. & Clin. Med. **35:** 8 (Jan.), 1950.

The discovery of the anticoagulant dicumarol led to the search for a 4-hydroxycoumarin with about the same hypoprothrombinemia-inducing capacity as dicumarol, but which also produced less toxic effects on small blood vessels. 4-hydroxycoumarin anticoagulant No. 63 was studied and was found to have an optimal effect in twenty-four to forty-eight hours; this is somewhat shorter than the period of optimal effect of a single dose of dicumarol. This drug still needs frequent prothrombin determinations as does dicumarol. From the authors' experience with dogs, 4-hydroxycoumarin anticoagulant No. 63 has a greater degree of anticoagulant action and a duration longer than that of dicumarol without the production of hemorrhagic manifestations. The potency of 4-hydroxycoumarin anticoagulant No. 63 is two to three times as great as that of dicumarol. The mechanism of action of this new drug has not been established, nor has it been precisely determined for dicumarol. Desired distinct advantages over dicumarol have not as yet been proven.

MINTZ

CONGENITAL ANOMALIES

Gasual, B. M., Richmond, J. B., and Krakower, C. A.: A Case of Tetralogy of Fallot with a Patent Foramen Ovale (Pentalogy) Showing a Marked Left Ventricular Hypertrophy and Left Axis Deviation. J. Pediat. **35:** 413 (Oct.) 1949.

The authors present clinical and pathological findings in a case of tetralogy of Fallot associated with patent foramen ovale. Of unusual interest was the finding of left axis deviation and hypertrophy of the left ventricle. It was concluded that the presence of the associated patency of the foramen ovale resulted in a shunt from the right to the left side of the heart in this case. The hemodynamics probably were very similar to those of tricuspid atresia or stenosis. The pressure in the right auricle must have been higher than that in the left auricle and part of the blood probably flowed from the right into the left auricle, while the remainder flowed into the right ventricle. The additonal blood flowing into the left auricle and thence into the left ventricle probably resulted in a

gradual hypertrophy and dilatation of both of these chambers. The right ventricle in this case was also hypertrophied because of the stenosis of the pulmonary valve and the dextraposition of the aorta. It is concluded that a patent foramen ovale in association with a tetralogy of Fallot may in some instances result in hypertrophy of the left ventricle sufficient to produce a left axis deviation in the electrocardiogram.

SCHWARTZ

Olim, C. B.: Coarctation of the Aorta at the Level of the Diaphragm. Ann. Surg. **130:** 1091 (Dec.), 1949.

The author presents a case of a young woman who at operation was found to have a coarctation of the aorta just above the diaphragm. She died five hours after surgery was performed. Preoperatively the blood pressure in the left arm was consistently found to be between 180/140 and 200/140, as compared with readings of 120/100 and 130/100 in the right arm. Blood pressure and pulsations were not obtainable in the lower extremities. Pulsations were not noticed over the intercostal arteries and there were only minimal erosions of the ribs. The aortic knob was present but not prominent.

ABRAMSON

Björk, S., and Liedholm, K.: The Femoral Pulse Curve in Coarctation of the Aorta. Acta med. Scandinav. **136:** 97 (Dec. 23), 1949.

The authors report what they believe to be a diagnostic and pathognomonic sign of coarctation of the aorta. In 3 patients a characteristic alteration of the femoral pulse curve was found. The peak of the curve is reached much later than normally. In patients with coarctation it is reached after mechanical systole while in normal subjects it occurs before the end of mechanical systole. In addition, there is a statistically significant delay in the peak of the carotid pulse curve. The secondary peak on the steep decline of the normal curve is absent in coarctation because of the absence of reflection of the pulse wave from the periphery.

Normally the femoral pulse wave is dependent on the pulse wave initiated by the stroke volume and propagated by the aorta. In coarctation the pulse wave is delayed and reduced at the constriction, so that in the femoral artery the pulse wave is formed not by the stroke volume but by fractions of it flowing through collateral channels into the distal part of the aorta at different moments and at different levels.

WAIFE

Ferguson, H., and Porter, C. E.: Complete Heart Block Associated with Pregnancy. South. M. J. **43:** 44 (Jan.), 1950.

Two cases of complete atrioventricular block associated with pregnancy are reported, one of them a patient who had had two pregnancies. None of the pregnancies resulted in cardiovascular complications. The cardiac diagnosis in each case was probable congenital heart block associated with a patent interventricular septum. These cases further substantiate the evidence that complete heart block is not in itself a contraindication to pregnancy.

LECLS

CONGESTIVE HEART FAILURE

Dagnini, G.: The effect of Prolonged Fast on the Hydro-Saline Metabolism of Patients in Congestive Failure. Folia cardiol. **8:** 207 (June), 1949.

Patients in congestive failure were submitted to a prolonged fast. If no edema was present, sodium chloride decreased in the blood and was excreted in the urine at a gradually decreasing rate. On the other hand, if the patients were edematous the sodium chloride of the blood did not vary; sodium chloride was excreted poorly at the beginning and then in greater amounts. This increased elimination paralleled an increased diuresis, so that the curve of elimination of salt and that of water were parallel. The total excretion of salt during fast in general is remarkable. The author believes that there is a temporary reduction of the sodium chloride in the blood and that passage of salt from the tissues to the blood causes reabsorption of the edema, and diuresis.

LUISADA

Barger, A. C., Richardson, G. S., and Roe, B. B.: A Method for Producing Chronic Cardiac Failure in Dogs. Proc. Soc. Exper. Biol. & Med. **73:** 113 (Jan.), 1950.

A form of progressive, chronic, "right-sided" cardiac failure was produced in dogs by tricuspid valve avulsion and pulmonary stenosis. Three dogs thus treated developed congestive failure with elevated auricular pressure and distended neck veins. The dogs also showed decreased work tolerance, hepatomegaly, ascites, tachycardia at rest and a relatively fixed heart rate during exercise. Two of the hearts studied in the heart-lung prepartion showed cardiac insufficiency.

MINTZ

Tepper, E.: The Effect of Strophanthin on Venous Pressure in Heart Failure. Deutsche med. Wchnschr. **75:** 142 (Jan.), 1950.

In three groups, consisting of patients without heart failure, with left heart failure and with right heart failure, venous pressure, arterial pressure, pulse rate and respiration were studied at one-minute intervals following an intravenous injection of 0.25 mg. strophanthin. In the patients without heart failure no changes of significance were observed. In both types of heart failure the pulse rate and the diastolic arterial pressure showed a slight decline. The respiration became slower and deeper

in left heart failure and the venous pressure increased markedly. In right heart failure the outstanding change was a drop of the increased venous pressure, the average fall being 50 mm. of water. The venous pressure began to fall one minute after the injection; this could also be observed in cases of latent or impending failure. A test injection of strophanthin is of value in the recognition of heart failure as well as in the differential diagnosis between right and left sided failure.

PICK

CORONARY ARTERY DISEASE AND MYOCARDIAL INFARCTION

Horlick, L., and Katz, L. N.: Retrogression of Atherosclerotic Lesions on Cessation of Cholesterol Feeding in the Chick. J. Lab. & Clin. Med. **34**: 1427 (Oct.), 1949.

The authors studied the effect of cessation of cholesterol feeding in chicks on the hypercholesterolemia and atherosclerosis which had been produced by such feeding. Within ten weeks after the procedure was terminated there was a gradual decline in the severity of the lesions, while blood cholesterol levels fell to normal within three weeks. Microscopically, there were found a diminution and disappearance of foam cell deposits, increasing fibrotic changes in both the abdominal and thoracic portions of the aorta, diminution of grossly stainable lipid and heavier calcification. In general, the early lesions were completely reabsorbed, while more severe lesions underwent regressive and reparative changes.

ABRAMSON

Brean, H. P., Marks, J. H., Sosman, M. C., and Schlesinger, M. J.: Massive Calcification in Infarcted Myocardium. Radiology **54**: 33 (Jan.), 1950.

The authors present 14 cases with calcification within myocardial infarcts. In eight of the cases calcification was visible in life. Such calcification is most likely to be noted within the fibrosed myocardium of patients with large myocardial infarctions who survive for six years or more. It is most frequent in the region of the cardiac apex and may be differentiated from pericardial calcification by its position within heart muscle rather than on the very contour of the silhouette. The authors stress that calcification of infarcted myocardium, as in ventricular aneurysm, occurs in individuals with a predominant right coronary artery pattern, or with a balanced pattern, but is infrequent when the chief source of blood supply is from the left coronary artery. The clinical course in these patients is the same as in other patients with similar grades of infarction who have no calcification.

SCHWEDEL

Habbe, J. E., and Wright, H. H.: Roentgenographic Detection of Coronary Arteriosclerosis. Am. J. Roentgenol. **63**: 50 (Jan.), 1950.

The authors describe a technic for the roentgenographic detection of calcified coronary arteries. Four spot films are taken on a 10 × 12 inch film. The first film, the posteroanterior view, is made with a 3-inch cone in close relationship to the posterior chest wall to the left of the spine at the atrioventricular junction; the second exposure is made with the patient turned 5 to 10 degrees into the right anterior oblique position, the cone being to the left of the spine; the third is made with the patient rotated about 5 to 10 degrees into the left anterior oblique position, the cone being to the left of the spine; the fourth, is made with the patient turned about 20 to 30 degrees into the left anterior oblique position, the rays being projected to the right of the spine. Rapid exposures, high (360) milliamperage, and the usual kilovoltage (60 to 70 KUP) are desirable. Kymography and roentgenoscopy are used chiefly as accessory aids. The demonstration of a parallel row of linear or punctate calcified material at the expected sites is sufficient for the diagnosis.

The authors have found 28 cases of calcified coronary arteries, only 4 of which had right coronary artery involvement. Roentgenkymograms at times were helpful in demonstrating calcific areas moving synchronously with heart motion.

SCHWEDEL

Morrison, L. M., and Gonzales, W. F.: Effect of Choline as a Lipotropic Agent in the Treatment of Human Coronary Atherosclerosis. Proc. Soc. Exper. Biol. & Med. **73**: 37 (Jan.), 1950.

In view of the encouraging lipotropic action of choline on atherosclerosis in experimental animals, the authors made a study of the lipotropic action of choline in proven human atherosclerosis. A group of 230 patients with proved acute myocardial infarction were studied. One-hundred fifteen of these patients served as controls. The other 115 patients who had had acute myocardial infarction were placed on choline therapy after discharge from the hospital. The dose of choline varied from 6 to 32 Gm. daily per individual depending on the patient's tolerance for the drug and the degree of hypercholesterolemia present. Of the 115 control patients, 35 patients or 30 per cent died within three years. In the choline-treated series of 115 patients, 14 patients or 12 per cent died after three years. Thus, over a three-year period the lipotropic agent choline was effective in reducing the mortality rate due to recurrent coronary thrombosis with myocardial infarction.

MINTZ

ELECTROCARDIOGRAPHY

Kilaidonis, P., Tilmant, J. Gouffault, J., Deschamps, H., and Carouso, G.: Contribution to the Study of Left Ventricular Hypertrophy. Arch. d. mal. du coeur **42**: 700 (July), 1949.

The authors studied 463 electrocardiograms significant of left ventricular hypertrophy. The tracings

were divided into several groups according to the etiology and the clinical form of heart failure. One group consisted of 67 cases with postmortem control.

The electrical axis of QRS was found most often in the first sextant of Bayley between 0 and minus 30 degrees. Deviations from this range occurred in arteriosclerotic heart disease, in aortic stenosis and in arterial hypertension. A right axis deviation was not found in the material. In congenital heart disease with left heart strain, the QRS axis was usually normal and A_{QRS} and A_T were infrequently opposite in direction. The electrical position was most frequently horizontal in all groups. A marked increase of the voltage in the standard leads was found if left ventricular hypertrophy was produced by aortic stenosis and insufficiency; there was less of an increase if the hypertrophy was due to arteriosclerotic heart disease. The same held true with the amplitude of S in V_1 and of R in V_6. In more than half of the cases the index of White and Bock exceeded or was equal to plus 17 mm. This index was normal in most of the patients with arteriosclerotic disease and with congenital anomalies. In one-third of the cases the intrinsicoid deflection in V_6 was delayed more than 0.45 sec. and in two-thirds of the cases it was associated with an augmented index of White and Bock. A QS complex in V_1 to V_4 was not indicative of anteroseptal infarction in the presence of signs of left ventricular hypertrophy. The most marked signs of left ventricular hypertrophy were found in cases of aortic stenosis.

PICK

Carouso, G., Deschamps, H., Tilmant, J., Lenegre, J., and Kilaidonis, P.: Electrical and Anatomical Study of Primary Mixed Cardiopathies. Arch. d. mal. du coeur. **42:** 711 (July), 1949.

The authors studied the electrocardiograms of 189 cases of "combined heart strain," including 148 cases of mitral-aortic valvular lesion, 14 cases of mitral stenosis with hypertension and 14 cases of hypertensive heart disease complicated by chronic pulmonary disease. Eighty-nine cases were in general heart failure. In 46 cases left heart failure was present, and in 15 cases, right heart failure. No signs of heart failure were found in the remaining 39 patients. Cases with bundle branch block and signs of myocardial infarction were excluded from the study. In 49 of the cases an autopsy control of the clinical data was available.

In their analysis of the electrocardiograms, the authors directed their attention to the presence of QRS-axis deviation and determination of the electrical position in the limb leads and to signs of rotation along the longitudinal axis and signs of right and/or left hypertrophy in the chest leads. The authors came to the following conclusions based on this analysis: In "mixed cardiopathies" (combined heart strain) the characteristic pattern in the limb leads consists of a QRS axis in the limb leads between 0 and +90 degrees, signs of clockwise rotation along the longitudinal axis and signs of left heart hypertrophy in the chest leads. In contrast to this pattern, pure mitral lesions lack the appearance of left heart strain in the chest leads, and cases with right heart hypertrophy secondary to left heart failure usually show left axis deviation in the limb leads and counterclockwise rotation in the chest leads.

PICK

Schaefer, H., and Trautwein, W.: On Elementary Electrical Processes in Cardiac Muscle and Its Role for a New Theory of the Electrocardiogram. Arch. f.d. ges. Physiol. **251:** 417 (Aug.), 1949.

The arrival of excitation at the exposed surface of the dog heart was measured by recording the electrical events on a double beam oscillograph by means of pairs of small differential electrodes. It was demonstrated that the endocardial breakthrough occurs simultaneously over various regions; this suggests that the conduction system supplies conducting twigs deep into the muscular layers of the myocardium. A region opposite the branching of the larger subdivision of the His bundle and located to the right of the interventricular sulcus was first to be activated in all experiments; this region is termed "source of excitation" (Quellpunkt der Erregung). The authors believe that small individual myocardial sections are selectively excited and that these regions are relatively isolated from each other during excitation, while recovery occurs simultaneously over much larger sections.

HECHT

Hanssen, P.: The Incidence of Auricular Flutter and Auricular Fibrillation Associated with Complete Auriculoventricular Dissociation. Acta med. Scandinav. **136:** 113 (Dec. 23), 1949.

Complete A-V dissociation was found in 66 patients among 59,000 admissions over a nine-year period. Auricular flutter was also present in 7 and auricular fibrillation in 8 subjects. There were 38 males and 28 females. Heart block was permanent in 64 per cent, chiefly in those over 60 years of age. Forty-two of the 66 had various degrees of the Adams-Stokes syndrome. This occurred more frequently in males but showed no particular age distribution. Eighteen per cent had angina pectoris; 6 per cent had myocardial infarction. Forty-four per cent had some degree of heart failure. Bundle branch block was noted in 55 per cent of the cases. There was no sex difference among the cases of complete heart block associated with auricular flutter or fibrillation. Heart failure with angina pectoris was more frequent in this group of 15 patients than in patients with complete heart block alone.

WAIPE

HYPERTENSION

Díaz, C. J., Barreda, P., and Molina, A.: The Mechanism of Nephrogene Hypertension. Rev. clin. espan., **35**: 375 (Dec. 31), 1949.

The authors report 2 cases of apparently "nephrogene" hypertension, in which decapsulation of the kidneys with denervation of the renal pedicle and splanchnic nerve resection resulted in profound hypotension and death in irreversible shock a few hours after the operation. They tried to simulate the mechanism which was active in these patients in animal experiments: In 5 dogs hypertension was produced by "cellophane nephritis" and two to nine months later the kidneys were removed. In all 5 cases a marked drop in blood pressure resulted, which lasted until the death of the animals. In control experiments on 8 normotensive dogs, bilateral nephrectomy brought about a rise in blood pressure up to hypertensive levels. The authors' conclusion is that the hypertensive effect emanating from the kidneys can be twofold: overproduction of renin or faulty production of hypertensinase. It might be difficult to decide in an individual case which is the actual mechanism, but the latter seems to be the more frequent.

ULLMANN

Maier, H. C.: Intrathoracic Pheochromocytoma with Hypertension. Ann. Surg. **130**: 1059 (Dec.), 1949.

The author reports a case of pheochromocytoma in which the tumor was located in the costovertebral area just lateral to the upper portion of the descending aorta. On removal of the mass, the systolic blood pressure dropped from 220 to 110 mm. Hg and a few minutes later was barely obtainable. With intravenous adrenalin, a gradual elevation of blood pressure occurred. For several years after operation the blood pressure ranged between 160/100 and 180/110 but later it fell to 140/80.

The author points out that a diagnosis of intrathoracic pheochromocytoma should be considered when a tumor in the costovertebral portion of the chest is associated with paroxysmal or sustained hypertension, especially in the younger age group.

ABRAMSON

Peet, M. M., Isberg, E. M., and Bassett, R. C.: Hypertension Complicated by Spontaneous Subarachnoid Hemorrhage. Am. J. Surg. **78**: 912 (Dec.), 1949.

Subarachnoid hemorrhage of nontraumatic origin may be the result of a ruptured intracranial arterial aneurysm, extension from a massive intracerebral hemorrhage, meningeal inflammation, bleeding from a neoplasm or a blood dyscrasia. A ruptured intracranial aneurysm is by far the most common cause.

During the acute phase of subarachnoid hemorrhage, the patient is strictly confined to bed with his head elevated, and therapy is entirely supportive. Lumbar puncture is performed for diagnosis only, with the amount of fluid slowly withdrawn being no more than 1 to 2 cc. After the acute stage has subsided, intracranial arteriography is performed and if an aneurysm is revealed which is readily accessible to surgical isolation, this is treated in an appropriate manner. Since the presence of hypertension increases the mortality from subarachnoid hemorrhage, the authors recommend splanchnicectomy in such instances in an attempt to produce a persistent, significant reduction in blood pressure. Through such means the possibility of recurrence of bleeding from another aneurysm is decreased.

ABRAMSON

Calkins, E., Dana, G. W., Seed, J. C., and Howard, J. E.: On Piperidylmethyl-Benzodioxane (933-F), Hypertension, and Pheochromocytoma. J. Clin. Endocrinol. **10**: 1 (Jan.), 1950.

The authors point out that 933-F "reverses" the hypertensive effects of epinephrine but has little effect on the hypertension caused by direct sympathetic stimulation; therefore this drug is of value in diagnosing pheochromocytomas, especially if associated with a sustained hypertension. Using an intravenous dose of 20 mg. of 933-F, they studied 120 patients with hypertension. In 2 patients with polycystic kidneys, 2 with hypertension of pregnancy, 2 with chronic pyelonephritis, and 1 each with chronic glomerulonephritis and Cushing's disease, none responded to 933-F with a fall in blood pressure; in fact 3, including the case with Cushing's disease, showed a rise. In only 2 out of the total group was there a significant fall in blood pressure, the others showing a negligible blood pressure change or a rise. Almost all patients developed a tachycardia and became flushed; occasionally premature systoles were encountered, and some had a fine tremor. Most patients complained of palpitation, nervousness and a sense of warmth.

Of the 2 patients with a positive response to 933-F one was found to have a pheochromocytoma at surgery. The other, a 7 year old female, had a neuroblastoma, removal of which caused a gradual fall in blood pressure, and a negative response to 933-F; with recurrence, however, the pressure rose again, and a positive response to 933-F reappeared. Small amounts of epinephrine-like substance were extracted from the tumor.

It is postulated that sometimes pheochromocytomas may produce hypertension indirectly by stimulating the pituitary and adrenal, rather than directly by the effect of their secretions on the arteriolar bed or cardiac musculature.

CORTELL

Handler, P., and Bernheim, F.: Importance of Dietary Protein, Calories and Salt in Experimental Renal Hypertension. Am. J. Physiol. **160**: 31 (Jan.), 1950.

Rats were made hypertensive by a figure eight

ligature of the left kidney and removal of the remaining kidney. This type of hypertension was influenced by at least three dietary factors: caloric intake and protein and sodium content of the diets. Restriction of food consumption adequate to permit weight maintenance resulted in a fall to normal pressure levels regardless of the amount of protein offered. Low protein diets also caused a significant reduction in hypertensive readings. Drastic reduction of sodium failed to lower pressures in high protein fed rats but caused a significant fall to subnormal levels in low protein fed rats. The addition of 3 per cent sodium chloride to a low protein diet resulted in marked hypertension.

HECHT

Greco, G. F.: Hypertensive Heart Disease In Older Patients. Geriatrics **5:** 15 (Jan.–Feb.), 1950.

In the older patient, hypertensive heart disease usually takes a benign course. The condition of the vascular tree and not the height of the pressure determines the progress of the disease. Early roentgen findings are a convex rounding of the left border of the heart due to left ventricular enlargement, and elongation of the silhouette, indicating left ventricular dilatation and involving chiefly the outflow tract extending from apex to aortic valve. The rounded apex is displaced to the left and down toward the chest wall, plunging posteriorly below the dome of the diaphragm. The high QRS voltage is due to the increased left ventricular mass, with an increased potential directed toward the left arm. ST-segment depression and T-wave inversion occur due to relative myocardial ischemia and to chemical changes affecting myocardial nutrition.

For successful management, the physician should become familiar with the personality of the hypertensive and the patient's entire life must be reviewed in relation to his social and economic goals. Sedation with bromides and phenobarbital, given routinely throughout the day, may lead to confusion, restlessness and disorientation. Chloral hydrate and paraldehyde are preferred.

WAIFE

Perera, G. A.: The Adrenal Cortex and Hypertension. Bull. New York Acad. Med. **26:** 75 (Feb.), 1950.

On the basis of observations in animals and in man, the author concludes that the adrenal cortex is related to hypertension. He reports that, in animals: (1) Bilateral adrenalectomy interfered with the development of experimental renal hypertension and also abolished or impaired renal capacity to form vaso-excitor materials under anaerobic conditions in vitro, even in animals maintained on high salt diets. (2) Large doses of pituitary adrenocorticotropic hormone at times stimulated the juxtaglomerular apparatus which may secrete a vasopressor material. (3) Desoxycorticosterone acetate (DCA) plus salt produced cardiac and renal enlargement in dogs and also caused hypertension in nephritic rats.

In human subjects, he found that: (1) Prolonged administration of DCA at times produced hypertention in patients with Addison's disease as well as in normotensive subjects without adrenal disease. (2) DCA, 10 mg. daily for one week, caused a prompt rise in blood pressure in hypertensive patients but not in normotensives. (3) Rigid restriction of sodium chloride masked the pressor response of hypertensives to DCA. (4) Cortisone, 80. mg. daily, caused small decreases in blood pressure in four hypertensive patients and an increase in one patient with Addison's disease.

WHITE

Green, D. M. and Peterson, E. M.: Hypertensive Encephalopathy after Administration of Benzodioxan. J. A. M. A. **142:** 408 (Feb. 11), 1950.

A 55 year old woman with paroxysmal hypertension, associated with attacks of blurred vision and right-sided convulsions, received 10 mg. of benzodioxan per square meter intravenously. Within six minutes her blood pressure rose from 168/96 to 190/110. This was associated with convulsive twitchings and thick speech. The attack subsided without residual signs after intravenous sodium amytal. This attack mimicked her previous attacks exactly. The cold pressor test and a histamine test did not provoke such a reaction. It is concluded that benzodioxan has a sympathomimetic action in a susceptible person.

WAIFE

PATHOLOGIC PHYSIOLOGY

Poli, G., and Rossi, C. R.: Influence of the Liver on Cardiac Activity. Arch. di fisiol. **48:** 143 (Jan. 20), 1950.

Experiments in which the livers of dogs were excluded from the circulation confirmed the known fact that this organ exerts a favorable action on the heart. When a liver was included in the circulation, or when hepatic blood was injected, both mean and pulse pressure increased. Regular, slow undulations of the blood pressure were observed after exclusion of the liver, while a steady level of the blood pressure was maintained after injection of hepatic blood. The above observed phenomena were not due to mechanical or chemical effect of the injected solution, as proved by several control studies.

The authors are still uncertain whether to admit a hepatic hormone or the existence of other actions. One possibility is that the liver removes from the blood some depressant metabolic product so that it can not reach the heart. In other words, the observed phenomena might be caused by toxic properties acquired by the plasma when the liver is not included in the circulation.

LUISADA

Rougier, G. and Cabanes, L.: Cardiac Output in High Altitude. Compt. rend. Soc. de biol. **113:** 185 (Sept.), 1949.

In order to determine the cardiac output in man under anoxemic conditions, the authors placed healthy individuals in a low-pressure chamber for half an hour and lowered the atmospheric pressure within twenty minutes to values corresponding to an altitude of 4000 meters. An increase of the cardiac output, determined by the ethyl iodide method, from 30 to 50 per cent was observed. In untrained persons this was due mainly to an increase of the heart rate, in trained individuals mainly to an increase of the stroke volume. Following exercise under anoxemic conditions a further increase up to 75 per cent occurred.

PICK

Maier, H. C.: Blood Stasis in the Lungs as a Factor in the Etiology of Postoperative Pulmonary Complications. Ann. Surg. **130:** 790 (Oct.), 1949.

The role of circulatory stasis in the lungs as an etiologic factor in postoperative pulmonary complications is emphasized by the author. Normally the lungs can accommodate large quantities of blood in their capillary beds without any increase in external dimensions. The increased volume of blood in the lungs causes encroachment on the alveolar spaces by the engorged capillaries. When the subject assumes the recumbent position, there is a reduction in vital capacity, since the lungs now serve as a storage place for the blood which has drained out of the veins in the lower extremities. Because of the muscular inactivity and depressed function of the surgical patient during and immediately after operation, there is a demand for less blood in large portions of the body. As a result, accumulation of blood occurs in the lungs. Abnormal respiratory excursions and lack of change in body position over a period of hours further contribute to circulatory stasis. The blood stasis particularly when present in the dependent parts of the lung, predisposes the postoperative patient to the development of atelectasis and pneumonia. More attention should therefore be directed to the avoidance of pulmonary blood stasis.

ABRAMSON

Gossels, C. L.: Auricular Fibrillation Caused by Electric Accident (Effects of Electric Current on the Human Body). Exper. Med. & Surg. **7:** 335 (Nov.), 1949.

The author reports the case of a 49 year old man previously in good health who touched the lower wires of a high tension power line with an aluminum ladder. He was instantly knocked to the ground and remained unconscious for several minutes. A short time later an examination revealed a ventricular rate of 130 with a barely palpable pulse and a blood pressure apparently too low to be recorded. Three hours after the accident the blood pressure was 80/60 and auricular fibrillation with ventricular premature beats was observed on the electrocardiogram. The pulse rate was 120. For the following two days the pulse and blood pressure remained in the same range. The patient was digitalized and on the following day (the third day after the accident) the electrocardiogram was normal. It was not until the fifth day that the blood pressure increased to 140/70. A slight tremor of the hands remained for a longer period of time.

A review of the literature by the author shows 7 reported cases of auricular fibrillation, or auricular flutter, after electrical accidents. The pathologic finding in electrical fatalities is intensive blood stasis. Anatomical lesions of the heart are rarely seen. Rupture of the myocardial fibers and fragmentation of the myocardium have been reported.

MARGOLIES

Akman, L. C., Silber, E. N., Miller, A. J., and Katz, L. N.: Repolarization in the Dog Ventricle: Effects of Heating and Cooling Entire Epicardial Surface. Am. J. Physiol., **159:** 492 (Dec.), 1949.

Cooling and warming the entire epicardial surface of the hearts of 7 dogs by perfusing the intact pericardial sac with isotonic saline solution produced opposite effects in the direction of the T wave in standard bipolar limb leads and in unipolar limb leads. Heating the epicardial surface caused increase in height of T; cooling resulted in inversion of T. S-T segment shifts occurred only at maximal temperature ranges, and the duration of the Q-T interval increased in direct proportion to the coldness of the perfusate. The authors conclude from these experiments that an endocardial-epicardial gradient exists during repolarization.

HECHT

McCann, W. S.: Some Neglected Aspects of Cardiology. Circulatory Function of the Respiration. Am. J. Med. **8:** 62 (Jan.), 1950.

The author describes the action of the lungs as a check-valve capable of regulating the transfer of blood from the right ventricle to the left; he thus seeks to clarify certain apparent discrepancies in the operation of Starling's law and the genesis of congestive failure of the circulation.

The extensive pulmonary capillary network receives blood from both ventricles through the pulmonary and bronchial arteries. If one pulmonary artery is ligated these capillaries will fill and become distended from the higher pressure bronchial arteries. The pressure within this capillary network is the result of intra-alveolar air pressure, pulmonary and bronchial arteriolar pressure and pulmonary venous pressure. The appearance of the lung of the guinea pig at simulated high altitude is identical with that of a dog after ligation of a pulmonary artery or of a man with congestive failure; all show engorgement

of the capillaries of the alveolar walls with thickening and encroachment upon the alveolar spaces. Low oxygen tensions have been proved to increase pulmonary artery pressure in man and experimental animals apparently as the result of local arteriolar and precapillary constriction, accompanied, in the experimental animal at least, by systemic and therefore bronchial artery dilatation. The author believes that the changes in the lungs in congestive failure are not passive and that they may depend almost entirely upon anoxia rather than on back pressure from the left atrium. He states that there are indications that certain mechanisms, such as bronchial constriction, pulmonary arteriolar constriction, venopressor tone, right heart tone and rate of diuresis, operate to cushion the effect of a sudden increase in blood volume, venous return and atrial pressure and thus protect the alveolar capillaries from rupture. He then constructs a theory as to the manner in which this check valve function of the lung takes part in the genesis of congestive failure of the circulation.

H. D. LEVINE

Wegria, R., Keating, R. P., Ward, H. P., Dreyfuss, F., Frank, C. W., and Blumenthal, M. R.: Effect of Auricular Fibrillation on the Coronary Blood Flow. Am. J. Physiol. **160:** 177 (Jan.), 1950.

Measurement of coronary blood flow in sixteen dogs through the left anterior descending coronary artery by means of an electromagnetic rotameter revealed that during the onset of electrically induced auricular fibrillation a sharp drop occurs in coronary flow which is returned to normal and better than normal levels within a few seconds. Upon cessation of the irregularity coronary flow increases and remains high for several minutes.

HECHT

PATHOLOGY

Levy, A. L., and Patterson, M. C.: Acute Serofibrinous Pericarditis of Undetermined Cause. A Study of Twenty-Seven Cases. Am. J. Med. **8:** 34 (Jan.), 1950.

The authors describe a group of 27 patients with chest pain, most of whom had a friction rub and enlargement of the roentgen-ray shadow of the heart. In these patients neither the clinical course nor subsequent prolonged observation (six months to sixteen years, average five and one-half years) established the existence of either rheumatic fever or myocardial infarction. In most cases the attack followed in the wake of an acute upper respiratory infection or an atypical pneumonia. The pain radiated in the majority of cases, was more often intermittent than continuous, and in most cases was aggravated by deep breathing, cough or movement of the body. Dyspnea was experienced by 14 patients and 5 of these required the erect posture for comfort. A pericardial friction rub was heard in 19 cases. In not a single instance were there signs of shock or cardiac insufficiency. Left pleural effusion was present in 14 cases, complicated by right pleural effusion in six. The course was variable in severity and duration but the prognosis for recovery was always good. The occurrence of fever, a friction rub or an increase in the sedimentation rate on the first day of the illness was helpful in differentiating this type of pericarditis from that type complicating myocardial infarction. Over a period of from two weeks to three months (average six weeks) the electrocardiogram showed distinctive changes in the RS-T segment and T waves without depressed RS-T segments, a Q pattern or prolongation of the P-R interval. Recurrences, milder than the original attacks, occurred 4 times in 3 cases. The antistreptolysin titer was generally not elevated but it was high in two patients from whose throats a hemolytic streptococcus was grown. Penicillin, streptomycin and sulfadiazine had no apparent beneficial effect on the pericarditis. There was no evidence that this condition is a precursor of constrictive pericarditis.

H. D. LEVINE

Gottlieb, C., and Peck, W. K.: Report of a Case of Rupture of the Thoracic Aorta. Am. J. Roentgenol. **63:** 63 (Jan.), 1950.

The authors report the clinical and autopsy findings of a case of rupture of a saccular aneurysm of the lower thoracic aorta. The presenting symptoms at the time of admission simulated an acute gastrointestinal perforation. There were physical and roentgenographic signs suggesting a pneumonic consolidation in the right lower chest, but successive thoracenteses indicated a moderate serosanguinous effusion, followed within a few days by the aspiration of a thick bloody fluid which did not clot. The patient died about six weeks after the onset of symptoms. The necropsy findings were those of pinpoint perforation into the right pleural space.

SCHWEDEL

PHARMACOLOGY

Salter, W. T., Sciarini, L. J., and Rubin, B.: Inotropic Synergism of Cardiac Glucosides with Calcium Acting on the Frog's Heart in Human Serum. J. Pharm. & Exper. Therap. **97:** 314 (Nov.), 1949.

The authors describe a method for measuring minute quantities of digitoxin and ouabain in the serum of both experimental animals and patients under digitalis therapy. The frog's heart was used as a biological indicator. The inotropic effects rather than the toxic effects were measured. The authors believe that previous reports of the rapid disappearance of digitalis glucosides from the serum are in error because the previous technics were too crude to detect small quantities. At low levels the evaluation can be made in terms of calcium ions, when pH and potassium are controlled.

The contractile response of the frog heart bathed with human serum was depressed by the addition of citrate. This depression was mitigated by the subsequent addition of ouabain or digitoxin. It was possible to transform the citrate values into equivalent calcium concentrations for the serum in question. When equilibrated with ouabain dissolved in human serum, the frog heart retained the inotropic effect after being transferred to Krebs-Hensleit solution. When human serum was diluted with calcium-free Krebs-Hensleit solution, the calcium-protein complex behaved as a calcium buffer. Twenty per cent human serum (two parts of serum to eight of calcium-free synthetic solution) had a calcium-ion concentration of approximately 0.54 mm. In 3 cases of congestive heart failure, one of which was treated with ouabain intravenously and 2 of which received digitoxin orally, the serum concentration remained elevated for over an hour. The higher figures for digitoxin reflected largely the lower potency of digitoxin as compared with ouabain.

GODFREY

Ruskin, A., and Johnson, J. E.: Cardio-Depressive Effects of Mercurial Diuretics. Cardio-Protective Value of BAL, Ascorbic Acid and Thiamin. Proc. Soc. Exper. Biol. & Med. **72:** 577 (Dec.), 1149.

The purposes of this study were to learn the specific effects of Mercuhydrin on conduction and electrical systole in the isolated rabbit heart; to compare its myocardial toxicity with that of other organic mercurials; and to evaluate the detoxifying effects of BAL, ascorbic acid and thiamin, used before and after the mercurial compounds.

Mercuhydrin caused prolonged atrioventricular and intraventricular conduction time, prolonged the electrical systole, and produced dropped beats and cardiac asystole. Preliminary BAL, ascorbic acid and thiamin, in descending order of efficacy, raised the blocking and cardiolethal dosages of Mercuhydrin. The same drugs given after the mercurial compound partially reversed the cardiac effects. Theophylline ethylene diamine and epinephrine exhibited lesser cardio-protective activity. Magnesium sulfate, niacin and riboflavin had no cardio-protective activity.

MINTZ

Ruskin, A., and Johnson, J. E.: Cardio-Depressive Effects of Thiomerin: Cardio-Protective Attempts with BAL, Ascorbic Acid and Thiamin. Proc. Soc. Exper. Biol. & Med. **72:** 572 (Dec.), 1949.

The purposes of this study were (1) to obtain comparative cardiotoxic and cardiolethal dosage levels of Thiomerin and (2) to determine the cardioprotective effects of BAL, ascorbic acid and thiamin used before and after Thiomerin in the isolated perfused rabbit heart.

Thiomerin prolonged atrioventricular and intraventricular conduction times, prolonged electrical systole, and produced transient ventricular ectopic beats and ventricular fibrillation. Large doses of Thiomerin produced marked cardiac slowing, asystole, cardiac dilatation and decreased contractility of the heart. To a much lesser extent than other mercurials, Thiomerin produced monophasic type S-T segment displacements, doming and inversion of the T wave. The cardiotoxicity of Thiomerin is less than 1/200 that of Mercuhydrin and 1/1000 that of Salyrgan, theophylline and Mercuzanthin. The cardiolethal ratios of Thiomerin to Mercuhydrin are less than 1:700; the ratio of Thiomerin to the other mercurials is 1:1,800.

When Thiomerin was preceded by BAL, approximately 2 times the usual cardiolethal dose was tolerated. BAL also reversed the prolonged atrioventricular and intraventricular conduction times. Ascorbic acid increased the cardiolethal doses of Mercuhydrin and Mercuzanthin but not of Thiomerin. The slight conduction effects of small doses of Thiomerin were often partly reversed by ascorbic acid. Thiamine protected against cardiolethal doses of Mercuhydrin but not against those of Thiomerin.

MINTZ

Knowlton, A. I., Loeb, E. N., Stoerk, H. C., and Seegal, B. C.: The Development of Hypertension and Nephritis in Normal and Adrenalectomized Rats Treated with Cortisone. Proc. Soc. Exper. Biol. & Med. **72:** 722 (Dec.), 1949.

This study was undertaken to determine the influence of cortisone upon the development of hypertension and nephritis in both intact and adrenalectomized rats. The administration of cortisone did not prevent the development of experimental cytotoxin serum nephritis in the rat. Moderate hypertension developed in nephritic rats with intact adrenal glands when cortisone was injected, whereas severe hypertension appeared in adrenalectomized nephritic rats similarly treated with cortisone. The nephritic rats rendered hypertensive by the administration of cortisone presented no greater histologic evidence of renal damage than did normotensive control nephritic animals.

MINTZ

Garb, S.: Inotropic Action of Epinephrine, Norepinephrine, and N-Isopropyl-Norepinephrine on Heart Muscle. Proc. Soc. Exper. Biol. & Med. **73:** 134 (Jan.), 1950.

Goldenberg and associates stated that "nor-epinephrine hypertension is due to an increase of total peripheral resistance, with no significant change, or even a drop in cardiac output, whereas epinephrine hypertension is the result of a significant increase of cardiac output." It is not clear which of the factors that determine cardiac output are responsible for the difference of response to epinephrine and norepinephrine. One factor which can be evaluated is the

effect of the two substances on the contractile force of myocardium.

The authors found that both dl-norepinephrine (Arterenol) and N-isopropyl-norepinephrine (Isuprel) exert an inotropic action on heart muscle which is at least as great as that produced by U.S.P. epinephrine. This report indicates that the differences in cardiac output which Goldenberg et al. reported are probably not due to a lack of inotropic effect by dl-norepinephrine.

MINTZ

Ch'en, J. S., and Freeman, S.: The Removal of Cations From Solutions and the Rat's Alimentary Canal by H-Form Resins. J. Lab. & Clin. Med. **35:** 99 (Jan.), 1950.

Experiments on the combining capacities of the cation exchange resins Nalcite HCR, Duolite C-3, and Amberlite IR indicate that Nalcite HCR is the most efficient as judged by the uptake of sodium, potassium and calcium from solution.

Sodium, potassium and calcium can be removed from the animal body in significant amounts by the ingestion of cation exchange resins. The relative affinity of the resin for different cations and the cation content of the diet both influence the extent to which various cations will be removed from the body by resin. The in vitro and in vivo uptake of sodium by H-form resins is suppressed by calcium and potassium ions. A high calcium diet markedly interferes with the uptake of potassium and sodium of the resin. It may be possible to remove selectively cations from the body by proper control of the cation content of the diet and the form and amount of ingested resin.

MINTZ

Wakim, K. G., Peters, G. A., and Horton, B. T.: The Effects of A New Sympatholytic Drug (Priscol) on the Peripheral Circulation in Man. J. Lab. & Clin. Med. **35:** 50 (Jan.), 1950.

The venous occlusion plethysmograph with a compensating spirometer recorder, the digital plethysmograph and potentiometric recording of the skin temperature over various regions of the body, including the fingers and toes, was used to study the effects of Priscol on the peripheral circulation in man. Intravenous administration of 50 mg. of Priscol in 2 cc. of solution within a period of two minutes produced a definite increase in blood flow in the forearms and legs, which lasted several hours. The systolic blood pressure increased an average of 6 mm. Hg and the diastolic decreased an average of 11 mm. These changes in blood pressure were transitory; within fifteen minutes of the administration of this drug, the blood pressure returned to the preinjection level. The heart rate increased an average of 26 beats per minute, and the rate returned to normal within fifteen minutes after the injection of the drug. There was a definite increase in the amplitude of the pulse even during the rapid heart rate produced by Priscol. Priscol produced the greatest increase in skin temperature over the lower extremities, especially the toes.

MINTZ

Barrow, J. G.: Treatment of Paroxysmal Supraventricular Tachycardia with Lanatoside C. Ann. Int. Med. **32:** 116 (Jan.), 1950.

Lanatoside C was given intravenously to 26 patients who had the clinical and electrocardiographic findings of paroxysmal supraventricular tachycardia, after carotid sinus and ocular pressure, gagging, and Valsalva maneuver had failed to terminate the arrhythmia. A period of 60 to 120 seconds was used for the injection and a total of 1.2 mg. of the drug was used as a routine procedure. If in 30 minutes the rhythm had not reverted to normal, 0.4 mg. more of the drug was given intravenously. Usually before a second dose of lanatoside C was given, carotid sinus and ocular pressure were tried again. In all the patients, normal rhythm was restored within one and one-half hours after administration of the preparation referred to above.

WENDKOS

PHYSICAL SIGNS

Alslev, J.: The Clinical Picture of the Dissecting Aneurysm of the Aorta. Deutsche med. Wchnschr. **74:** 1422 (Nov.), 1949.

Basing his discussion on 4 observations with postmortem controls, the author describes the clinical features of dissecting aneurysm of the aorta with special reference to the differential diagnosis between it and acute coronary occlusion. The radiation of the pain, which may be excruciating, sometimes follows the course of the aorta. Restlessness, singultus and thirst may be observed here more often than in myocardial infarction. Pre-existent hypertension may persist up to twelve hours after the onset of the attack. Anuria may be observed as a result of dissection of the renal arteries or of shock. In one case an acute abdominal picture was present with unusual bradycardia. In the only case in which the correct diagnosis was made during life there was a marked anemia, extensive cutaneous hematomata, abdominal distention and a paraplegia of the lower extremities.

PICK

PHYSIOLOGY

Page, I. H., Taylor, R. D., and Prince, Ralph: Noradrenalin-like Substance in Blood Demonstration by Cross-Circulation. Am. J. Physiol. **159:** 440 (Dec.), 1949.

Cross circulation experiments in dogs demonstrated the presence of a normally occurring pressor substance in the blood of normal dogs and of dogs whose liver, kidneys, spleen or adrenal glands had

been removed. The blood of dogs with experimental renal hypertension seemed to contain greater amounts of the substance than normal controls but its occurrence was not sufficiently regular to suggest that the substance played a part in the maintenance of this type of hypertension. The indicator dogs, into which 600 to 1000 ml. of heparinized dog blood was transfused, were made highly sensitive by either nephrectomy or spinal cord section from C-6 caudad with carotid sinus denervation, or both. Pressure rises of 50 to 75 mm. Hg were observed during the infusion lasting from five to seven minutes with or without the donor dog in the circuit.

It is assumed that the pressor substance has many of the characteristics of norepinephrine because methods which sensitize norepinephrine enhance the blood pressure response, adrenergic blocking agents (Dibenamine and Pricoline) abolish the effects, and because infused norepinephrine, in contrast to epinephrine, can be cross-circulated with similar effects. Perfused blood alone, therefore, appears to be capable of exhibiting vasoactive properties.

HECHT

Juergensen, E.: Posture and Circulation. Deutsche med. Wchnschr. **74:** 1530 (Dec.), 1949.

The author observed a high incidence of liver enlargement in two groups of workers: wine growers and dental surgeons. Both occupations require protracted and strenuous work in a more or less fixed upright position. This leads, in the author's opinion, to an interference with the normal liver circulation and to a general circulatory disturbance, of which liver enlargement is the earliest sign.

PICK

Holldack, K.: The Significance of the Physiology of the Human Ear in the Daily Practice of Auscultation. Deutsche med. Wchnschr. **74:** 1497 (Dec.) 1949.

The diagnostic possibilities of auscultation have limitations due to the fact that the human ear can distinguish sounds and murmurs only within a certain range of intensity, frequency and time sequence. Thus, the differentiation of systolic and diastolic (presystolic) murmurs is possible only if they begin at least 0.1 sec. before or after the first heart sound. The quality of the first sound depends on this anatomical state of the mitral valve. Calcification of the leafflets leads to a decrease of its intensity. A loud first sound is due to a "misplacement" of the leaflets at the beginning of systole, and is found with a short A-V conduction time or with incomplete filling of the left ventricle in mitral stenosis or in shock. Additional (gallop) sounds cannot always be distinguished with certainty. A triple heart rhythm heard only during inspiration indicates splitting of a heart sound. A true gallop sound has a deep character. If the latter is heard together with a loud first sound, it indicates mitral stenosis even if no diastolic murmur is present. This combination is found only if thickening of the mitral leaflets is confined to the free border. Only such cases are suitable for a surgical approach to the valvular lesion.

PICK

Kempf, J. E., and Chang, H. T.: The Cardiac Output and Circulation Time of Ferrets. Proc. Soc. Exper. Biol. & Med. **72:** 711 (Dec.), 1949.

The ferret has been used as a laboratory animal for the study of numerous viral infections because of its susceptibility to these diseases. Diseases studied have been influenza, fowl-pox, swineherd's disease, Rift Valley fever, poliomyelitis, canine distemper and lymphocytic choriomeningitis. The ferret, because of its larger size, might serve better than the mouse or other rodents for investigations of these problems. Since little is known of the normal physiology of this animal, the present study was undertaken to study its cardiovascular function.

The mean cardiac output per minute per square meter of body surface for a group of 10 normal ferrets was found to be 2.476 liters. The average circulation time by the fluorescein technic was 6.8 seconds and 4.5 seconds as determined by the cyanide method. The average mean arterial blood pressure was 147 mm. Hg.

MINTZ

RHEUMATIC FEVER

Wedlum, B. G., Darley, W., Rhodes, P. H.: Prevalence of Rheumatic Heart Disease at High Altitudes. Am. J. Dis. Child. **70:** 205 (Feb.), 1950.

Colorado has been among the first five states with large death rates from rheumatic heart disease among school children for a period of over twenty years. For this reason, a study was conducted to determine the prevalence and severity of rheumatic heart disease at high altitudes. A total of 1017 children living at an altitude of 10,000 feet or above were examined. Ten children were found to have rheumatic heart disease, an incidence of 1 per cent, and seven had possible rheumatic heart disease. The authors conclude that rheumatic heart disease is not unusually prevalent or severe in children living at an altitude of 10,000 feet or above.

MARGOLIES

ROENTGENOLOGY

Fuchs, G.: The X-ray Differential Diagnosis of the "Small Heart." Wien. med. Wchnschr. **99:** 535 (Nov.), 1949.

Two types of the so-called "small heart" should be distinguished. The first type, found in asthenic but otherwise normal persons, is associated with a narrow thoracic cage and appears small by x-ray examinations because of its vertical position. The true small heart is more horizontal and is due to a congenital hypoplasia or may develop in dehydration

or as a consequence of chronic cachectic processes. The author reports one such observation on a patient with lymphosarcoma, in whom a cardiothoracic ratio of 1:2.6 was found and whose heart was placed horizontally within the thorax. At autopsy the heart was very small and showed marked brown atrophy of the myocardium.

PICK

Broden, B., Jonsson, G., and Karnell, J.: Thoracic Aortography. Observations on Technical Problems Connected with the Method and Various Risks Involved in Its Use. Acta radiol. **32:** 498, 1949.

The authors discuss the problems associated with the injection of a contrast medium into the aorta through a catheter inserted into the radial artery and by way of a cannula inserted into the common carotid artery. Their discussion is based on observations of 21 cases of aortic coarctation and 15 cases of patent ductus arteriosus.

In performing thoracic aortography with a catheter inserted with the right radial artery they met with the problems of arterial spasm, difficulty in directing the catheter into the aorta, and in bringing the tip of the catheter into correct position, especially into the ascending aorta. In the case of aortography with a cannula they stress that the outer of the double cannula must be inserted deeply enough to reach the aorta. To lessen cerebral complications (hemiparesis, convulsions) they suggest the injection of 50 cc. of 50 per cent rather than 70 per cent of organic iodine. The dye is injected with a specially constructed pressure apparatus through the catheter or the cannula, in from three to five seconds. Anteroposterior and lateral view roentgenograms are taken simultaneously.

SCHWEDEL

Jonsson, G., Broden, B., and Karnell, J.: Selective Angiocardiography. Acta radiol. **32:** 486, 1949.

The authors describe a method of angiocardiography in which the opaque dye is injected through a ureteral catheter (8F–9F). By selecting the site of the catheter tip various portions of the right atrium and right ventricle may be visualized. Apparently no better visualization of the left-sided chambers is obtained by injecting the dye into the pulmonary artery, except that there are no overlying densities due to residual dye in the right heart chambers. Demonstration of infundibular and pulmonic valvular abnormalities is usually better than with the older technics.

SCHWEDEL

SURGERY IN HEART AND VASCULAR SYSTEM

Ray, B. S., and Console, A. D.: Evaluation of Total Sympathectomy. Ann. Surg. **130:** 652 (Oct.), 1949.

The authors present the results obtained in 30 hypertensive patients subjected to total paravertebral sympathectomy, extending from the stellate to the third lumbar ganglia. Two in the series died as a result of the operation.

A quantitative relationship was found to exist between the extent of the sympathectomy and the degree of lowering of the blood pressure; this appeared to be dependent upon the more widespread vasomotor paralysis which followed total sympathectomy. However, the percentage of improved results in the blood pressure lowering effect was not great enough to justify the somewhat greater morbidity and mortality which attend the total sympathectomy, as compared with a lesser operation such as the thoracolumbar, T-7 or T-8 to L-3, and the splanchnic nerve resection. The advantages of total sympathectomy were that, since the stellate ganglia were removed bilaterally, there was an abolition or improvement of anginal pain because of interruption of afferent nerves that traverse the sympathetics, and also a slowing of the heart rate because of interruption of the accelerator nerves. Despite the extent of the operation, there was a partial return of tonus, probably due to the presence and augmented activity of residual sympathetic nerves which do not pass through the paravertebral ganglionated chains, the splanchnic nerves, or the celiac ganglia.

ABRAMSON

Babcock, W. W.: Aortic Aneurysm. South. M. J. **43:** (Jan.), 1950.

Most aortic aneurysms are due to degenerative arterial disease, which renders them less amenable to a direct surgical attack and more subject to fatal rupture than the traumatic aneurysms that involve the extremities. Operations that slow the blood current in the aorta increase the pressure upon the wall of the artery and favor rupture of the aneurysm even though the arterial obstruction is proximal to the aneurysm. The author describes an operation for decreasing the blood pressure within the aorta and aneurysm which has given relief from pain and greater longevity than the other operations used. In this operation it is essential that a large artery connected with the aorta be used and united, only end-to-end, to an adjacent vein in the direction of normal vascular flow.

LECKS

THROMBOEMBOLIC PHENOMENA

Kravchick, D. I., and Sheiman, L.: Evaluation of an in Vitro Heparin Tolerance Test for Thromboembolic Disease. J. Lab. & Clin. Med. **34:** 1222 (Sept.), 1949.

This work was based on the need for a laboratory test to predict thromboembolic disease. Waugh and Ruddick measured the ability of whole blood to counteract the anticoagulant effects of heparin in vitro. The basis for this test was the physiologic antagonism between thromboplastin and heparin; the former substance was considered to be increased

in the bloodstream in cases of accelerated coagulation. By using increasing amounts of heparin in a series of test tubes, they attempted a "controlled delaration of the clotting process." Silverman modified this test in an attempt to increase its accuracy. The authors used the Silverman modification in this report.

The in vitro tolerance test was studied in 74 controls, 12 obstetric patients, and 7 patients who had undergone prostatectomy. The authors found that this test failed to show a significant difference between the control and the post-partum–post-operative groups. These results differ from those in the reports of Waugh and Ruddick and Silverman.

MINTZ

Voorhees, A. B., and Pulaski, E. J.: The Fibrinogen B Test and Intramuscular Thrombosis. J. Lab. & Clin. Med. **34:** 1352 (Oct.), 1949.

Cummine and Lyons introduced the concept of an intermediary product in the fibrinogen to fibrin reaction to which they gave the name fibrinogen B. If fibrinogen B is found in the circulating plasma it is considered abnormal. They correlated its presence in the plasma with the production of intramuscular thrombosis. They also noted that fibrinogen B appeared in the plasma whenever tissue necrosis was present, especially in cases of pyogenic infection. Lyons also demonstrated in vitro that platelet fragility increases in the presence of fibrinogen B.

The authors made 553 tests and in only two instances were the criteria of Cummine and Lyons for inevitable thrombosis fulfilled, but neither patient developed clinical evidence of intravascular thrombosis. On the basis of this experience, the test would appear to be of no particular value in the confirmation of a diagnosis of thrombophlebitis suspected clinically. The authors were in agreement with Cummine and Lyons, however, in noting a relation between the presence of tissue necrosis and the appearance of fibrinogen B in the plasma. They also noted, as did Cummine and Lyons, a persistently negative test for fibrinogen B in thrombophlebitis after it was initially positive.

In view of the good results of anticoagulant therapy in intramuscular thrombosis initiated on clinical evidence alone, the practicality of this test is open to question.

MINTZ

Miley, G. P., and Dunning, P. M.: Ultraviolet Blood Irradiation Therapy (Knott Technic) in Thrombophlebitis. Am. J. Surg. **78:** 892 (Dec.), 1949.

Following the use of ultraviolet blood irradiation in 10 cases of acute deep thrombophlebitis, the authors noted a rapid disappearance of pain, tenderness along the course of the involved vein, and fever. Edema remained for from one to twelve days after treatment was started. The therapy also appeared to be effective in 9 patients with chronic thrombophlebitis. The procedure consisted of removing a predetermined amount of blood from an antecubital vein (1.5 cc. per pound of body weight) and passing it through the Knott hemo-irradiator which exposed the citrated blood at a definite rate to ultraviolet rays from a water-cooled quartz mercury vapor lamp. The irradiated blood was then injected back into the patient.

ABRAMSON

Hamlin, E., Jr., Warren, R., and Kennard, H. E.: Thromboangiitis Obliterans. An Evaluation of Therapy, with Special Reference to Lumbar Sympathectomy. New England J. Med. **241:** 849 (Dec.), 1949.

An attempt was made to evaluate sympathectomy as a therapeutic procedure in thromboangiitis obliterans. In a series of 85 patients, 170 extremities were sympathectomized. In 3 of 25 limbs intermittent claudication disappeared following operation, in 4 it became much less and in 8 it improved, while in the remaining 10 the symptom was unchanged or became worse. Most of the patients demonstrated improvement in general comfort. Twenty-three of the sympathectomized extremities were subsequently amputated.

ABRAMSON

James, T. G. I.: Thrombosis of the Internal Carotid Artery. Brit. M. J. No. 4639: 1264 (Dec.), 1949.

On the basis of a study of 3 cases of thrombosis of the internal carotid artery, the author calls attention to the value of carotid angiography in making the diagnosis. The branches of the external carotid artery are visualized but the internal carotid will show only in the first one or two centimeters of its course. Rarely the thrombosis may extend down to the origin from the common carotid.

It is pointed out that careful examination of the arteries of the neck in all patients with hemiplegia, convulsive states, or syncope may lead to a more frequent diagnosis of thrombosis of the carotid arteries.

Generally there is a sudden onset of hemiplegia, preceded by headache and dizziness. The arm is usually more affected than the leg and face. Hemianopia and hemianesthesia may be present and also aphasia, when the appropriate side is affected. Although some degree of recovery generally takes place, it is far from complete.

The etiology of the condition is not clear. Trauma does not appear to play any role. However, arteriosclerosis has been noted in a considerable number of cases and thromboangiitis obliterans in several.

ABRAMSON

Ameli, N. O., and Ashby, D. W.: Non-traumatic Thrombosis of the Carotid Artery. Lancet **2:** 1078 (Dec. 10), 1949.

The findings observed in 6 cases of non-traumatic

thrombosis of the carotid artery, as demonstrated by angiography, are reported. The first symptom in this condition is often some transient weakness of one of the extremities together with a minor disturbance of speech. After partial recovery takes place, several recurrent exacerbations of weakness and disturbance of speech occur, which then are followed by increased weakness, aphasia, loss of consciousness, or visual defects. In some instances slow improvement is noted, although in most cases the condition remains stationary for years. Angiography is the most important diagnostic measure. The radiopaque substance is injected into the common carotid artery. The commonest site of thrombosis is either at the point of bifurcation of the common carotid artery or a short distance above this site. Treatment does not differ from that of hemiparesis in cerebral thrombosis. The use of anticoagulants is contraindicated because of the possibility of cerebral hemorrhage.

ABRAMSON

VASCULAR DISEASE

Steindl, H.: Evaluation of the Different Therapeutic Procedures in Arterial Vascular Diseases of the Lower Extremities. Wien. med. Wchnschr. **99:** 530 (Nov.), 1949.

The author presents a survey of various surgical procedures for the management of arterial diseases in the lower extremities. He stresses the importance of early treatment and recommends repeated intra-arterial injections of a combination of acetylcholine (0.2 Gm.) with Prostigmine (0.5 Gm.) dissolved in a 1 per cent solution of procaine. Under this treatment he noted marked subjective and functional improvement due to an increase of blood supply to the afflicted extremity as evidenced by a rise of the skin temperature in the injected area. This was especially true in patients with a vasospastic component. Lumbar sympathectomy may result in permanent improvement whereas paravertebral anesthesia (L-2 to L-5) has only a temporary effect, but is useful for instantaneous relief of pain and for a quick demarcation of beginning gangrene.

PICK

Bartmann, K., Krautwald, A., and Voelpel, W.: Examination of the Vascular Function of the Lower Extremities by Novocaine Blockade of the Sympathetic Chain and of the Posterior Tibial Nerve. Ztschr. f. ges. Inn. Med. **4:** 713 (Dec.), 1949.

Both paravertebral sympathetic blockade and anesthesia of the posterior tibial nerve are useful for the establishment of the vascular function of the lower extremities in the ambulatory patient, since neither of them involves the nerve supply to muscles necessary for walking. In order to establish a standard method the operation was performed on 16 healthy persons between 24 and 57 years of age. Elimination of the vasoconstrictor tone of the foot was determined by thermoelectric measurements of the skin temperature of the dorsal and plantar side of the foot. An evaluation of 17 experiments revealed that paravertebral anesthesia is not suitable as a standard method; the results are inconsistent and the procedure difficult, dangerous and unpleasant for the patient. This is in contrast to anesthesia of the posterior tibial nerve. With the latter procedure, performed at a constant room temperature of 24 C., a skin temperature in the examined area of less than 35 C. is abnormal and values below 35.25 C. suggestive of inadequate blood supply to the leg. At a room temperature of 20 C. the respective abnormal and suggestive values are 34.00 C. and 34.45 C.

PICK

Wagner, F. B., Jr., and Herbut, P. A.: Etiology of Primary Varicose Veins. Histologic Study of One Hundred Sapheno-femoral Junctions. Am. J. Surg. **78:** 876 (Dec.), 1949.

In an attempt to study the factors involved in the production of primary varicose veins in the lower extremities, histologic sections were made of the saphenofemoral junction in 100 subjects. In only 16 cases was the sinus wall of the saphenous vein opposite the valve normal. In all others the sinus was from one-half to one-fifth of the normal thickness, as a result of absence of smooth muscle and internal elastic membrane in this site. The question was raised as to whether this defect was congenital and possibly hereditary. The valve leaflets were histologically normal in all instances.

ABRAMSON

Lyall, D.: Reactions following Injections of Varicose Veins. Am. J. Surg. **78:** 909 (Dec.), 1949.

The author states that patients exhibiting allergies of any sort should be tested for sensitivity to sclerosing solutions before injection of varicose veins is attempted. At times patients who have had injection therapy in the past may acquire sensitivity when a long period is allowed to elapse between injections. If too much sclerosing agent is used at any one time, the material may actually erode the vessel wall and cause a sterile abscess. Excessive dosage may also result in pigmented spots or streaks to a greater degree than necessary and may cause severe pain and fever. Patients with such clinical vascular disorders as thrombophlebitis, Raynaud's disease, Buerger's disease, vasospasm, and arteriosclerosis obliterans should not be subjected to injections of sclerosing agents, since the added spasm consequent to their use may reduce the caliber of the involved vessels or their collateral bed to a critical degree and thus contribute to the production of gangrene. Successful sclerosis involves the use of a minimum

amount of material and the subsequent maintenance of the vein in a collapsed state, through the prolonged use of elastic bandages or stockings, until healing occurs.

ABRAMSON

Le Fèvre, F.: Physical Medicine and Peripheral Vascular Disease. Arch. Phys. Med. **30:** 774 (Dec.), 1949.

The author emphasizes the importance of physical medicine as therapy in peripheral vascular disorders. He points out that the application of controlled heat may be of value in certain conditions, although the use of uncontrolled heat may cause great harm, particularly in conditions in which arterial insufficiency exists. Frequently night cramps, which are not specific for vascular disease, will be helped by an electric blanket. Vascular exercises and the oscillating bed may reduce the rest pain of occlusive arterial vascular disease and may also decrease the edema of the legs which is often present in this type of condition. Whirlpool baths cause relaxation of muscles and are comforting, although they do not affect intermittent claudication. They seem to be especially effective in the treatment of varicose ulcers and secondary stasis dermititis. Pressure suction boot treatment and intermittent venous hyperemia appear to have little, if any, role as therapy in peripheral vascular disorders.

ABRAMSON

Gibson, P. C.: Aspirin in the Treatment of Vascular Diseases. Lancet **2:** 1172 (Dec. 24), 1949.

Since aspirin is considered to act as an anticoagulant at the same time that it relieves pain, the author used the drug in anginal pain and in venous thrombosis. The dosage adopted was similar to that utilized in rheumatic carditis. According to the author, the results produced sufficient alleviation of symptoms to warrant further trial with the drug.

ABRAMSON

Fowler, E. P., Jr.: Capillary Circulation with Changes in Sympathetic Activity. I. Blood Sludge from Sympathetic Stimulation. Proc. Soc. Exper. Biol. & Med. **72:** 592 (Dec.), 1949.

Stimulation of the cervical sympathetic trunk and the application of local epinephrine or norepinephrine to the conjunctivae and the injection of epinephrine intravenously has produced the picture of decreased circulation from narrowing of the vascular bed. It also produces what looks like "sludging" of blood within the vessels. Intravenous procaine in small doses will often reverse the sludging process, at least for a time, but this varies with the concentration of novocaine and the speed of injection.

MINTZ

MISCELLANEOUS

Radner, S.: Subclavian Angiography by Arterial Catheterization. Visualization of Metastatic Tumor in the Upper Thoracic Aperture. Acta radiol. **32:** 359, 1949.

The author describes a technic of catheterizing arteries of the upper thorax and neck wherein the radial artery is exposed under local anesthesia, incised, and a ureteral catheter (6F–9F) is inserted and guided upwards into the subclavian artery under fluoroscopic control. Fifteen cc. of a radiopaque dye are injected rapidly, and roentgenograms are taken immediately after injection and three to four seconds later. Compression of the brachial artery with a sphygmomanometer prevents the flow of the dye peripherally.

The author feels that this method is suitable for investigation of structures supplied by the subclavian artery, such as the parathyroid glands, parts of the thyroid gland and the thymus. An illustrative case is described in which a metastatic tumor in the upper thoracic aperture was visualized by this method. After the injection of the dye the brachial artery is ligated above the incision site. The author states that no untoward effects have resulted from the entire procedure.

SCHWEDEL

Elam, J. O., Neville, J. F., Jr., Sleator, W., and Elam, W. N., Jr.: Source of Error in Oximetry. Ann. Surg. **130:** 755 (Oct.), 1949.

The oximeter is a photoelectric device for continuous determination of arterial oxygen saturation, which depends upon the differences in light absorption by the pigments oxyhemoglobin and reduced hemoglobin. The model that can be obtained commercially is applied to the ear lobe, the blood in the ear first being arterialized through the application of heat. According to the authors, the clinical value of the oximeter justifies its further development. However, certain precautions must be taken, particularly with regard to the means of arterializing the ear blood and the way in which the 2 photocells are mounted in the earpiece. The development of a quantitative oximeter is a long range program which still requires considerably more work and improvements in technic.

ABRAMSON

Boone, B. R., Ellinger, G. F., and Gillic, F. G.: Electrokymography of the Heart and Great Vessels: Principles and Application. Ann. Int. Med. **31:** 1030 (Dec.), 1949.

This instrument has proved a valuable tool in the field of cardiovascular physiology but its value in clinical work remains to be defined. Promising results have been obtained in preliminary studies of the arrythmias and myocardial infarcts. In the latter

condition, it has been possible to demonstrate clearly paradoxical movement of the infarcted ventricular wall. However, in examinations being done on a large group of older men, several instances of paradoxical type movement in subjects with no or minimal cardiovascular symptoms and a normal electrocardiogram have been encountered without any apparent cause. Records from subjects with valvular heart lesions have been difficult to analyze. One case diagnosed aneurysm of the thoracic aorta and another diagnosed mediastinal tumor by the electrokymograph have been verified at surgery and by response to x-ray therapy, respectively. It remains to be determined in what specific types of cardiovascular disease pathognomonic electrokymographic patterns appear and just what clinical value the instrument will have.

WENDKOS

BOOK REVIEWS

Quinidine in Disorders of the Heart. *Harry Gold, M.D.* New York, Paul B. Hoeber, Inc., 1950. 100 pages, $2.00.

Gold's monograph is the first book dealing solely with the use of the important drug, quinidine. The author has discussed in turn the details of indications, therapeutic actions, toxic actions, effect on the electrocardiogram, clinical pharmacology, dosage, and the prevention and treatment of all the minor and major arrhythmias with which the practitioner may be confronted. There is a very brief discussion of the use of quinidine in children and of alternative routes of administration, and a chapter on combined use of quinidine and digitalis. The section on pharmacology of the drug is clear and concise, as would be expected from the author's special qualifications. The reviewer strongly concurs in Gold's statement that "the use of inflexible systems of dosage is responsible for a large share of the defeats in quinidine therapy, and some of the disasters." Gold touches upon, but does not emphasize as clearly, that the disasters are often due as much to failure of careful supervision, with repeated clinical and electrocardiographic observations during and after conversion of an arrhythmia, as to the inflexibility of the system of dosage.

The major criticism that the reviewer has of the monograph concerns Gold's handling of the chapter on chronic auricular fibrillation. Many cardiologists would disagree with his statement (on page 53) that the use of quinidine in longstanding auricular fibrillation has, for the most part, been abandoned. He admits that quinidine is credited with accidents which are purely coincidental and stresses the fact of occasional serious complications of quinidine therapy without giving sufficient emphasis to the hazards of auricular fibrillation, per se, and to the benefits that may be obtained by converting some patients with auricular fibrillation to normal rhythm.

One might question the author's recommendation to avoid the simultaneous use of quinidine and digitalis whenever possible. His stress on the toxicity of quinidine in animals poisoned with digitalis does not do justice to the clinical fact that the two drugs have been repeatedly used in combination without difficulty when poisoning with either drug is avoided. If quinidine is used without prior digitalization in patients with auricular fibrillation, acceleration of the ventricular rate with quinidine may be so great as to force cessation of quinidine therapy.

With the reservations noted in the discussion of chronic auricular fibrillation and of the combined use of digitalis and quinidine, this monograph can be strongly recommended to the practitioner as an excellent guide to the understanding and successful use of quinidine.

MAURICE SOKOLOW

Digitalis and other Cardiotonic Drugs, ed. 2. *Eli Roden Movitt, M.D.* New York, Oxford University Press, 1949. 245 pages, 20 figures, 11 tables, $5.75.

The author presents a review of the clinical use of digitalis preparations. Divided into chapters dealing with digitalis folium, digitalis lanata, lanatoside C, other digitalis glycosides (including digitoxin, Digoxin, Gitalin), strophanthus, squill and other cardiotonic agents of plant origin, the book, in addition to a brief historical sketch, reviews the most recent literature on the clinical use of each digitalis preparation. The recent literature on the mechanism of action of digitalis is well reviewed. The bibliography at the end of each chapter is extensive. For the general practitioner who wishes to appreciate the volume of work at present under investigation in digitalis therapy, this book is excellent. The book is, however, lacking in critical analysis of much of the work done by others which is incorporated in the text. Impres-

sion are accepted as well as actual data. It is apparent that none of the author's own experience is represented, although he may express an occasional opinion which differs or confirms the results reported by others. Unfortunately, too much emphasis is placed upon results of individual digitalis preparations rather than the similarity of the fundamental principles involved for all preparations for the treatment of cardiac disorders.

ROBERT C. BATTERMAN

Morbus Caeruleus. An Analysis of 114 Cases of Congenital Heart Disease with Cyanosis. *E. Mannheimer, ed.* Basel and New York, S. Karger, 1949. 332 pages, 122 figures, 34 tables, $9.25.

It is important to remember that this book is not intended as a text but as an integrated report of diagnostic and therapeutic procedures in congenital heart disease with cyanosis. To this purpose it includes a careful analysis of clinical, roentgenologic, angiocardiographic, electrocardiographic, and physiologic studies, as well as general diagnostic and surgical summaries and 42 complete case reports. A comprehensive bibliography adds to the reference value of this publication. From the viewpoint of clinical usefulness the type of material presented will probably prove most valuable to those already experienced in the diagnostic problems of congenital heart disease. To the inexperienced, the statistical data with its variations from any central tendency of the group may be confusing when an attempt is made to apply these data to an individual case.

Observations of fundamental importance are made, particularly in the sections on roentgenography and electrocardiography, but some of the conclusions based on these observations may not find general acceptance. Supported by the evidence of proved cases, the basically important point is made that "in a great many cases it is very difficult to determine by x-ray the absolute and relative sizes of the right and left ventricles." The clinical implications of such a statement are of very great significance. This point is further amplified in a demonstration of the comparative value of multiple unipolar precordial leads as contrasted with the limitations of the conventional extremity leads. In this connection, tracings are shown which display other than the type of ventricular enlargement expected from the degree of right or left axis deviation present in the standard lead electrocardiogram. Another important x-ray observation is the variety of heart sizes and contours which may be compatible with the diagnosis of uncomplicated tetralogy of Fallot. The supplementary value of angiocardiography is also well demonstrated. All of the x-ray and angiocardiographic reproductions together with explanatory line drawings are of excellent quality.

The physiologic studies reported are of interest but do not add significantly to fundamental knowledge on the subject which has been established largely by others to whom proper credit is given. The results of the hypoxia tolerance test are new and indicate the remarkable adaptation of children with congenital heart disease and cyanosis to chronic anoxia. Further studies on circulation time are necessary to supplement the results indicated by these authors.

No publication written at this time on the subject of congenital heart disease can be expected to be free of unchanging opinions or, frequently, gross mistakes. This is probably inevitable, not because of inadequacies on the part of the investigator, but because of the early stage of the development of knowledge on the subject. Taking this into consideration it may be said that the work reported by Mannheimer and associates is well done and well written, and the opinions expressed are stimulating and valuable if not always absolutely correct. This book represents about the only concerted effort to correlate all of the various types of studies on such a group of patients, and, if for no other reason, on this basis alone it should prove valuable to all interested in the subject of congenital heart disease with cyanosis.

ROBERT F. ZIEGLER

Congenital Anomalies of the Heart and Great Vessels: Clinicopathologic Study of 132 Cases. *Thomas J. Dry, Jesse E. Edwards, Robert L. Parker, Howard B. Burchell, H. Milton Rogers, and Arthur H. Bulbulian.* With a foreword by Dr. F. A. Willius. Springfield, Charles C Thomas, 1948. 68 pages, 96 figures, $4.50.

Our knowledge of congenital heart disease is today undergoing profound changes as a result of surgical advances and the application of angiocardiography and cardiac catheterization. Until recently studies of congenital heart disease have been mainly anatomical, and attempts at physiological and clinical correlation have not been too successful. With the advent of physiologic approaches, the field is at present in a state of confusion and this confusion will not be entirely dispersed until anatomy and physiology are wed, which is the problem of the future. It is only human that we think only in terms of today and forget the great names of the past and their fundamental contributions on which our current knowledge and future knowledge depend.

The appearance of this book is a timely reminder of our heritage. The authors represent the fields of medical history, internal medicine, cardiology—both clinical and physiological—, anatomy and pathological anatomy at the Mayo Clinic. The contents of the book were originally prepared as an exhibit at the Centennial Meeting of the American Medical Association in June, 1947. It is a clear, concise, clinicopathologic study of 132 cases of congenital heart disease collected over a twenty-five-year period. Sixteen representative specimens of the common congenital abnormalities are described. Photo-

graphs of 16 of the key figures in the field of congenital heart disease introduce each case report. A thumbnail sketch of the contribution of each of the 16 (William Hunter, Thomas Bevill Peacock, William Harvey, Jean-Baptiste De Sénac, Franklin Paine Mall, James Hope, Henri-Louis Roger, Victor Eisenmenger, Etienne-Louis Arthur Fallot, Eduard Sandifort, Karl Rokitansky, Sir Arthur Keith, G. A. Gibson, Julius Tandler, John Baptist Morgagni, Maude E. Abbott) is included.

Each defect is taken from an autopsied case with a brief account of the history and physical findings. The essential anatomy and the path taken by the blood is described and illustrated by a colored drawing, a photograph of the actual specimen, and a diagrammatic sketch. The sketches are to be commended for their simplicity and clarity. A posteroanterior roentgenogram and an electrocardiogram of the patient in question are included. The principal clinical features of each anomaly are enumerated and the incidence of the anomaly in this series is noted. A generous bibliography is appended. The arrangements conform to the usual high quality of the publishers.

As Dr. Willius states in the foreword, "It is . . . an opportune time for the authors of this monograph . . . to make their work, and the work of others, available to the medical profession in this manner. Their years of experience in this field, their own investigations, and the long hours of application required to complete this work, have produced an undertaking of great merit and usefulness." To those who are concerned with the problems of congenital heart disease, this monograph is recommended not as a reference or as a text so much as a milestone linking the past with the present and future.

LEWIS DEXTER

Lehrbuch der Röntgenologischen Differentialdiagnostik. Erkrankungen der Brustorgane, ed. 2. *Werner Teschendorf.* Stuttgart, Georg Thieme, 1950. 779 pages, 865 figures, $21.00.

This is the second edition of a work first published in 1939. There has been considerable editing with deletion of several pages of the old text and the addition of a few new references and illustrations to replace the old ones. This review deals with the cardiovascular material scattered throughout this general text on the roentgenology of the thoracic contents, and includes the pulmonary manifestations of heart disease.

About 20 pages are devoted to various types of heart measurements, five to cardiac contours in the posteroanterior and both oblique positions, and about 50 pages to typical configurations in various disease states. The bulk of the section specifically devoted to the heart is concerned with examples of the use of conventional multiple slit kymography. Some of the illustrations of calcification of coronary arteries and of the mitral and aortic valves are rather well depicted; the portions on pericardial effusion and calcification are discussed at length but rather obliquely so that the exact meaning of some of the observations is obscure.

Vague and unsubstantiated statements are scattered throughout the section on the heart; for example, statements that myocardial weakness is indicated by low voltage QRS complex in the electrocardiogram combined with diminished amplitude of pulsations on kymography, and that contour configurations indicate specific combinations of entities. The manner of presentation is old fashioned, heavy-handed Teutonic dissertation, usually without mention of proof. The following is the literal translation of an example: "the question concerns a prominent pulmonary artery in a 41 year old female suffering from bronchial congestion since childhood. The physical examination was negative, no murmurs. There was a right axis deviation on the electrocardiogram. The roentgenologic differential diagnosis lies between patent ductus arteriosus or a congenital pulmonary artery lesion. Syphilitic disease of the pulmonary artery must be excluded. The possibility exists that the dilatation was caused by the closure of a previously open ductus arteriosus." This example leaves one completely uncertain as to what the diagnosis actually was since there was no anatomic diagnosis, no clinical diagnosis; only the roentgenologic one.

Such presentations served their usefulness in the early days of roentgenology but are wholly inadequate today. To publish a book in 1950 on roentgenological diagnosis without even mentioning angiocardiography and electrokymography is hardly excusable. The mere editing of a previous edition is not a substantial basis for the issue of another printing as a second edition.

JOHN B. SCHWEDEL

Electrocardiography. *Louis Wolff.* Philadelphia, W. B. Saunders, 1950. 182 pages, 110 figures, $4.50.

Part I of this book (79 pages) is devoted to "the basic principles of electrocardiography," and Part II (99 pages) deals with "clinical electrocardiography." Although Part I is a lucid, orderly discussion of basic principles, orientation is probably insufficient to provide the uninitiated with a basis for analysis of the wave form of the electrocardiogram. The solid angle, the axis of injury, and the vectorcardiogram are not introduced, and the mean axis receives little more than definition. It is gratifying to observe that nearly one-half of the text is devoted to basic principles. The division of the text into two parts is unfortunate, however, for it perpetuates the dictum that basic principles are something apart from clinical electrocardiography.

Part II contains a well organized description of carefully selected curve patterns covering the normal, bundle branch block, hypertrophy, myocardial infarction, short P-R interval syndrome, and pul-

monary embolism. Care is taken to differentiate the electrical from the clinical diagnosis. Experienced electrocardiographers will object to the occurrence of inaccurate, though easy-reading, statements on the grounds that the beginner may be mislead rather than helped by them. Examples are (page 41) "The form of the [unipolar limb lead] curve recorded . . . will depend on the part of the heart that the limb root faces, or looks at," and (page 96) "Inverted T waves in left ventricular curves of right bundle branch block are not normal, ..." Nevertheless, the text is highly recommended for beginners and for "empiricists" who find themselves imbedded in a maze of unclassified unipolar leads.

ROBERT H. BAYLEY

Physiology in Health and Disease, ed. 5. *Carl J. Wiggers.* Philadelphia, Lea and Febiger, 1949. 1242 pages, 280 figures, $10.00.

The appearance of the fifth edition of Wiggers' textbook of physiology needs no introduction to those interested in the cardiovascular field. For many years his writings have left an indelible impression on all working in the field. The present revision has satisfactorily kept abreast of the recent advances in experiment and in concept in a number of intensively active fields, including hypertension, artificial kidneys, dyspnea and aviation physiology. A readable chapter on the physiologic basis for psychosomatic reactions serves to introduce the student to clinical problems which at first sight appear to be entirely outside the realm of basic sciences. The section on the heart and circulation represents a working summary of the author's major interest over several decades and remains an outstanding, if not *the* outstanding, presentation of the subjects. The treatment of hemodynamics, cardiac action and the electrical activity of the heart, while basic, is geared to an explanation of clinical findings. Students, investigators and clinicians alike will continue to find the volume of considerable assistance in helping them to review and revise their understanding of physiologic processes which are basic in health and disease.

LOUIS N. KATZ

Flebotrombosis y Embolia Pulmonar (Phlebothrombosis and Pulmonary Embolism). *Francisco Rojas Villegas.* Santiago, Chile, Imprenta "Rome," 1948. 221 pages, 17 figures, 13 charts.

This monograph on the important problem of phlebothrombosis and pulmonary embolism is a welcome addition to the Spanish medical literature on the subject because of its completeness and extensive bibliography. It is a pity, however, that the plates of the monograph are so devoid of relative meaning because of the lack of clarity of detail.

The author considers the etiology, pathologic physiology, symptoms and signs of phlebothrombosis and pulmonary embolism with descriptions of important diagnostic procedures, one of which is the x-ray demonstration of the point of occlusion by means of phlebograms. This is illustrated by the author in several cases but with plates which have been reproduced poorly. This procedure has not gained favor in this country except by Ochsner and his group, because of the difficulty of interpretation and because of the danger of injection of substances for visualization of the veins. The author states: "this method should be practiced whenever it is possible so that one may gain a personal experience of the technic and the interpretation of the x-ray films and be in a position to take advantage of this experience when it is necessary." This is certainly a radical point of view, and this reviewer believes that more emphasis should be placed on clinical findings rather than to a procedure such as phlebography.

The fifth chapter on treatment and prevention is adequate except for the rather short section on heparin-dicumarol therapy and its merit relative to surgical ligation of the femoral and external iliac veins and the vena cava. Of course, the author's own series is too small for any statistical evaluation of the relative merits of these procedures. The problem is one which is still debatable. Should all cardiovascular cases who will have many bouts of failure and phlebothrombosis before their death have ligations? Or should we reserve anticoagulant therapy for postoperative patients with incidental thrombosis? Recent papers have stressed the fact that anticoagulant therapy is at least as good if not better than surgical ligation, taken as an over-all picture.

Even though the book does not have any new thoughts on the subject, it will serve to focus the attention of the Latin physician on this important problem.

BERNARD E. NUNEZ

Die Elektrokardiographie, ed. 4. *A. Weber.* Berlin, Springer, 1948. 209 pages, 150 figures

This fourth edition of a well-known book, which, according to the author, was ready for print in 1943, discusses electrocardiography, as well as registration of heart sounds, of respiration, of the cardiogram and of the venous pulse. As in the previous editions, the discussion of the physical basis for these registration methods and the description of the different apparatus comprises a considerable portion of the book and is one of its advantages. The ventricular complex in the electrocardiogram is explained by the summation of two monophasic currents only. The tracings which represent right or left ventricular strain patterns are explained by a delayed conduction in the right or left bundle branch, respectively. The disturbances of cardiac rhythm are discussed in the conventional way. The figures are well reproduced. This monograph will be of value to those readers who wish to familiarize themselves with conceptions of the genesis of the normal and of the hypertrophy electrocar-

diograms which are so different from our own. Chest leads are given little consideration.

DAVID SCHERF

La Spartéine en thérapeutique. *Fernand Mercier.* Paris, Masson et Cie, 1948. 103 pages, 25 figures, 165 francs.

This is an attempt to revive interest in sparteine, now seldom if ever used in this country. Evidence (much of it obtained by the author and his associates) is cited to indicate that sparteine has valuable effects on the heart (combining the useful actions of digitalis, quinidine and atropine), the autonomic nervous system (a nicotinic block limited to the parasympathetic division, without demonstrable stimulant phase), the central nervous system (sedative, anesthetic and anticonvulsant), voluntary muscle (curare-like), uterus (oxytocic, capable of inducing or accelerating labor), and kidney (increased excretion of water and salt). Furthermore sparteine is said to be capable of protecting animals against otherwise fatal doses of snake venom or anaphylactogen. The evidence is insufficient to overcome the contrary findings of other workers or to call for reconsideration of the current American views on sparteine.

CARL F. SCHMIDT

The Heart and the School Child. A Symposium. *Jacob M. Cahan, ed.* New York, Tab Printing Corporation, 1949. 94 pages, $2.50.

This is an excellent little book on a most important subject. Its main shortcoming is the lack of discussion about what to do for the child in whom heart disease has been recognized. The diagnosis has been made, he is over his active rheumatic fever, he is to go back to school: how much can he do? This question faces every family doctor. Too often children with pathologic murmurs are needlessly curtailed in their activities, and yet the conscientious doctor is frequently at loss to know what advice to give. More adequate discussion of this subject could have made this book even more interesting and useful than it already is.

THOMAS W. CLARK

Diseases of the Heart. *C. K. Friedberg.* Philadelphia, W. B. Saunders, 1949. 1081 pages, 79 figures, $11.50.

This excellent book contains extremely comprehensive discussions of all phases of diseases of the heart, purposely omitting peripheral vascular diseases. Its most outstanding characteristic is the clear and thorough review of current concepts of circulatory dynamics and of the pathogenesis of heart diseases. These are not only the major content of the first 10 chapters, which collectively would be a valuable monograph, but likewise are continually woven into the discussions of the numerous clinical pictures presented. The literature, including material very recently published, is critically covered by an extensive and applicable bibliography which, with a competent alphabetical index, makes this an admirable reference work.

The writing is clear and well edited, but in attempting to be thorough and emphatic some space seems to be wasted in repetition. Perhaps some space might have been saved by discussing valve defects and their mechanical consequences under the etiologic entity in which they most commonly occur.

No attempt is made to provide the fundamental principles of electrocardiography, so that it is assumed that this information will be acquired elsewhere prior to consulting the numerous references to electrocardiographic findings in specific entities. Although most references to electrocardiography in such a wide group of diseases necessarily antedates 1944, it is hoped that later editions will permit inclusion of terminology and data largely based on current unipolar technics. This is particularly the case in electrical axis, hypertrophy and Q_1T_1 and Q_3T_3 myocardial infarction patterns.

Certain opinions of necessity must be the author's and are bound to precipitate controversy, but the book would not be a vital expression if these comments were excluded. Treatment of syphilitic heart disease and contraindications to quinidine therapy are examples.

It would seem preferable, once the concept of myocardial ischemia is presented on a quantitative basis as cause for angina pectoris, prolonged spontaneous cardiac pain, and myocardial infarction of varying severity, that it not be split into the several separate chapters on angina pectoris and myocardial infarction. The chapters on rheumatic fever and myocardial infarction, are specific examples of thorough and critical exposition, but all sections maintain a high degree of excellence in presentation of practical clinical diagnosis and therapy as well as pathogenesis and disturbed physiologic states of the circulatory system.

This book is a fine contribution to the current literature on heart disease and is worthy of broad acceptance by the practitioner seeking clear specific discussions of a particular cardiac disease, by the student as a textbook, both in pathologic physiology and the clinical phases of heart disease, and by those seeking a thorough and efficiently prepared reference source.

JOHN J. SAMPSON

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ANNUAL ELECTIONS

Dr. Howard B. Sprague, of Brookline, Mass., a former President of the New England Heart Association, was elected President of the American Heart Association for the 1950–51 term at the Twenty-Sixth Annual Meeting held last month at the Fairmont Hotel, San Francisco. Dr. Sprague, who is Clinical Associate in Medicine at the Harvard Medical School, succeeds Dr. H. M. Marvin of New Haven, Conn.

Dr. Louis N. Katz of Michael Reese Hospital, Chicago, was chosen President-Elect for the 1951–52 term. Dr. Katz is a member of the Association's Board of Directors and Chairman of the Research Committee of the Scientific Council. Other officers elected were Dr. Maurice Visscher, Minneapolis, Minn., Vice President; and Grant Keehn, New York, N. Y., Treasurer.

At a meeting of the Board of Directors, A. W. Robertson, Pittsburgh, was re-elected Chairman. Dr. John J. Sampson, San Francisco, was elected Secretary, and Dr. Harry Ungerleider was elected Assistant Secretary of the Association.

New members of the Board of Directors include: Edgar V. Allen, M.D., Rochester, Minn.; Emmet B. Bay, M.D., Chicago, Ill.; Ralph L. De Groff, Baltimore, Md.; Edgar Durbin, M.D., Denver, Col.; Frank N. Isbey, Detroit, Mich.; Ernest B. MacNaughton, Portland, Ore.; Augustus Martin, New York, N. Y.; Don G. Mitchell, Summit, N. J.; Sylvester L. Weaver, New York, N. Y.

The following were re-elected to the Board: E. Cowles Andrus, M.D., Baltimore, Md.; William H. Bunn, M.D., Youngstown, Ohio; S. DeWitt Clough, Chicago, Ill.; Robert L. King, M.D., Seattle, Wash.; Mrs. Wendell Kinney, Los Angeles, Calif.; Robert L. Mehornay, Sr., Kansas City, Mo.; David D. Rutstein, M.D., Boston, Mass.; John J. Sampson, M.D., San Francisco, Calif.

Representatives on the Board from the component councils and sections of the Scientific Council include:

Alva Bradley, Cleveland, Ohio; Julian S. Butterworth, M.D., New York, N. Y.; T. Duckett Jones, M.D., New York, N. Y.; Thomas M. Durant, M.D., Philadelphia, Pa.; R. Bruce Logue, M.D., Atlanta, Ga.; Hugh McCulloch, M.D., Chicago, Ill.; H. M. Marvin, M.D., New Haven, Conn.; John R. Smith, M.D., St. Louis, Mo.; Robert D. Taylor, M.D., Cleveland, Ohio; Robert W. Wilkins, M.D., Boston, Mass.; Leigh Willard, New York, N. Y.; Irving S. Wright, M.D., New York, N. Y.

Officers of the Scientific Council are: Chairman, H. M. Marvin, M.D., New Haven, Conn.; Vice-Chairman, Frank N. Wilson, M.D., Ann Arbor, Mich.; and Secretary, Lowell A. Rantz, M.D., San Francisco, Calif.

GOLD HEART AWARDS

Gold Heart Awards were presented at the Association's Annual Dinner last month for outstanding contributions in the field of cardiovascular diseases. These went to the four surviving signers of the Association's original articles of incorporation:

Dr. Lewis A. Conner, New York, first President of the Association and first editor of the American Heart Journal, the Association's former official publication; Dr. Robert H. Halsey, New York, President of the Association in 1931–32; Dr. Hugh McCulloch, Chicago, a member of the Executive Committee of the American Council on Rheumatic Fever; and Dr. Paul D. White, Boston, President of the Association in 1940–41, Executive Director of the National Heart Advisory Council, and Clinical Professor of Medicine at Harvard University.

In addition, awards were presented to Alva Bradley, Cleveland, for his outstanding work in securing the interest of corporations in sup-

porting heart research, and Mrs. Louise Baer, New York, for her outstanding contribution in securing general public support. Mr. Bradley, a realtor and former owner of the Cleveland Indians, is now Chairman of the American Council on High Blood Pressure Research. Mrs. Baer, wife of the columnist, Arthur "Bugs" Baer, is a Board member of the New York Heart Association and served as Chairman of New York's 1950 campaign.

CAREER INVESTIGATORS

Career investigatorships are being established as part of the Association's program to support the continuing careers of able investigators in the cardiovascular field. A brochure describing the requirements is available on request. Nominations for Career Investigators may be made by members of the Association's Scientific Council, deans of medical schools, and heads of research units in the United States. Nominations should be sent to Dr. Charles A. R. Connor, Medical Director of the Association.

PUBLIC HEALTH COMMITTEE

A Special Advisory Committee of authorities in public health, preventive medicine and social work has been formed to assist the Association's Public Health Division in working out specific activities for community cardiovascular disease programs. Dr. William A. Brumfield, Jr., Deputy Commissioner of the New York State Department of Health, was named Chairman of the committee. Other members include: Martin Cherkasky, M.D., Home Care Executive, Department of Home Care, Montefiore Hospital, New York; Ward Darley, M.D., Dean of School of Medicine, University of Colorado; W. Thurber Fales, Sc.D., Director, Statistical Division, Baltimore City Department of Health; Frederick G. Gillick, M.D., Chief, Heart Disease Control Branch, Division of Chronic Disease, U. S. Public Health Service; Leonard J. Goldwater, M.D., Professor of Industrial Hygiene, School of Public Health, Columbia University; Ira V. Hiscock, Sc.D., Chairman of Department of Public Health, School of Medicine, Yale University; E. A. Irvin, M.D., Medical Director, Cadillac Motor Co., Detroit, Michigan; Charles T. Mears, Executive Director, Pennsylvania Heart Association; H. M. Marvin, M.D., Associate Clinical Professor of Medicine, Yale University; Dorothy B. Nyswander, Ph.D., Professor of Public Health Education, School of Public Health, University of California; Leo Price, M.D., Director, Union Health Center, New York; David D. Rutstein, M.D., Professor of Preventive Medicine, School of Medicine, Harvard University; Miss Bessie Schless, Director of Social Service, Beth Israel Hospital, Boston, Mass.; Miss Margaret B. Taylor, R.N., Associate Professor, School of Public Health, University of Minnesota; James L. Wilson, M.D., Professor of Pediatrics, University of Michigan.

PROCEEDINGS OF CARDIOVASCULAR CONFERENCE

The complete proceedings of the first National Conference on Cardiovascular Diseases held in Washington January 18–20 are now available in book form. Copies may be obtained from the Association at $1.75 each by writing to Dr. John W. Ferree, Public Health Director. The volume includes the full text of reports by the Sections on Technical Knowledge and Research, Community Services, and Professional Education. These reports present comprehensive information regarding the present state of knowledge in the cardiovascular field, proposals for applying this knowledge more effectively, and suggestions for further study in certain areas.